THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. XXXI JUNE, 1934 No. 6

THE SPECIFICITY OF PULMONARY CONSOLIDATION IN TUBERCULOUS PATIENTS (EPITUBERCULOSIS)

RESOLUTION OF EXPERIMENTAL TUBERCULOUS PNEUMONIA*

By HENRY STUART WILLIS
NORTHVILLE, MICHIGAN

IT IS a common experience to those who care for tuberculous patients to observe in them the development of pneumonic consolidation which involves a comparatively large area of a lobe or even an entire lobe. This is particularly prone to appear in children. The question arises perennially as to whether such areas of disease are specifically tuberculous in nature or whether they have other origin. Most observers have come to view these as being manifestations of tuberculosis but numerous writers attribute various causes to them. It is the general impression that these consolidated areas occur definitely more frequently than would be expected if they were caused by the pneumococcus, i.e., that they are encountered more commonly in tuberculous patients than they are in the population in general.

Recent work[1,14,15,19] has brought out most of the salient facts in the literature. However, it may be stated briefly that usually the consolidated areas under consideration have been attributed to one of the following several causes: atelectasis, edema, pneumococcus pneumonia, nonspecific pneumonia, nonspecific pneumonia in tuberculous individuals and known as epituberculosis, and tuberculous pneumonia. The actual etiology has long remained a controversial matter.

Concerning atelectasis, there remains much of fact to be added to our knowledge before we are justified in speaking on this subject as glibly as we do at present. The term is used by some to mean one thing, by others to mean something else. Some restrict its use to the clinical entity of massive collapse or complete (and usually rapidly developing) atelectasis of a lobe or a lung. Others consider it simply to mean airlessness and thus they would have it apply to any diseased area of the lung which contains no air; this would include pneumonia, many areas of tuberculosis and numerous other conditions. Still others reckon the term to apply to the collapse of any part of a lung that is physiologically or functionally capable of expansion, that is, areas which are not diseased. The histologist tells us that terminal air passages are end-systems which have no intra-alveolar communication with adjoining structures of other end-systems and that thus local atelectasis is possible. Miller[12] has indicated the ease with which slight stretching or slightly rough manipulation

* From the Wm. H. Maybury Sanatorium (Detroit Municipal Tuberculosis Sanatorium), Northville, Michigan.

of pulmonary tissue produces artefacts that look like pores. It is his belief that demonstrable intra-alveolar apertures or communicating ways do not exist normally, although they may be present in pneumonia and other pathological conditions.

On the other hand, certain physiologists and experimental pathologists, notably Van Allen and Lindskog[20] have demonstrated functional if not anatomical intra-alveolar connections or "pores" through which air passes quite readily, and have thus indicated that atelectasis must be lobar in extent when it exists. They assert that there can be no true atelectasis short of that involving an entire lobe unless it be brought about by diseased alveolar walls which act as barriers to the air in neighboring air spaces. If local atelectasis is proved to be nonexistent, this phenomenon cannot explain the local or partially lobar character of the consolidated areas under consideration. Another fact against this contention is this, that physical examination is usually quite different in pneumonic consolidation and massive collapse and is highly characteristic in each condition. In massive collapse the physical examination and roentgen ray reveal retraction of the affected side, elevation of the diaphragm and displacement of the mediastinal structures toward the side of the disease.

As to edema as a cause of the roentgenological appearance under review, it may be said briefly that the very highly developed lymph drainage apparatus of the lungs, with abundant anastomoses in its vast network, the localized nature of many of these consolidated areas, the manner of clearing, the roentgen appearance of known pulmonary edema, and the physical signs all indicate that it has little to do with the formation of these characteristic shadows. Several of these and other possible etiological factors have been well reviewed and considered very recently by Reichle.[15]

Pneumococcus pneumonia occurs in tuberculous patients but it does so only rarely in the patients who live the relatively protected and sheltered life which the sanatorium offers. And the diagnosis is usually not difficult, with the characteristic onset, sputum, bacteriological findings, course and crisis. The pneumococcus may lead to lobar or bronchopneumonia in tuberculous individuals, but in no pneumococcus infection does the illness run the long, protracted course with the relatively few symptoms that characterize the typical consolidation in the tuberculous.

That a peculiar, nonspecific pneumonic consolidation may occur in tuberculous individuals (and in these only) is the now well-known contention of Eliasberg and Neuland,[5] who gave the affection the name "epituberculosis." The only criteria for ordinary pneumonia which these consolidated areas offer are the physical signs of consolidation. On the other hand, it seems clear that they offer several points which make the diagnosis of tuberculosis highly probable if not mandatory. These features are: (1) the long duration, (2) the lack of stormy symptoms, (3) the necessary presence (according to the authors' definition) of tuberculous infection as evidenced by the tuberculin reaction, (4) the presence of tubercle bacilli in the sputum as demonstrated in numerous instances, (5) the histological appearance of excised pulmonary tissue in a clinical case, and (6) the reappearance of the condition in children after injection of tuberculin.

The specificity of this type of pneumonic consolidation cannot seriously be doubted in the face of the last three facts just mentioned, as brief comment upon each will show.

(4) As a rule, tubercle bacilli are wanting in these cases but in one of our patients, cited as illustrative, they were present for a brief period during the height of the consolidation but were absent on repeated sputum examinations before and after this event.

This patient was a child, aged five, whose father had died from tuberculosis two weeks before our first contact with the child. This

contact revealed a marked reaction to the Pirquet test and signs of extensive tuberculosis of the right lung and the region of the root. While a patient in the sanatorium during the next year and a half, he grew normally and experienced marked absorption of the pulmonary infiltration so that there were only a few linear shadows in the region formerly diseased. At this time the tracheobronchial lymph nodes had become distinctly enlarged. Five months later, i.e., two years after detection, while still an inmate, the boy began to feel badly. His temperature rose occasionally to 101° F. and his pulse was accelerated, and in a six weeks' period he lost the 10 pounds he had gained during his

FIG. 1. Roentgenogram of the lung of a five year old boy showing an area of consolidation near the root of the right lung. This process was considerably more extensive five months ago but the film is not available.

stay. At this time he showed physical and roentgenological signs of massive consolidation in the same region of the right lung that had previously been involved. On one occasion during the time when the consolidation was pronounced, the sputum contained tubercle bacilli but it was negative before and has been negative since. The lesion subsided more quickly than is usual in "epituberculosis." The film taken three months later showed marked resorption and the one now (two and a half years later) shows little besides a few linear shadows. The child is in apparent good health. The films

FIG. 2. Roentgenogram of the same boy one year later. There has been marked clearing of the pulmonary lesion, associated with distinct increase in shadows in the mediastinum from enlargement of the tracheobronchial lymph nodes.

illustrating various stages of this child's disease appear as Figures 1 to 5.

(5) An almost compelling bit of evidence for the specificity of the condition has been given by Rubinstein[17] who examined pulmonary tissue removed by biopsy from a

FIG. 3. Film of the same boy taken nearly seven months after that shown in Figure 2. It reveals a marked recrudescence of the disease in the right lung at the former site. This was not present three months before this film was taken. Tubercle bacilli were present in the sputum at this time.

of pulmonary tissue produces artefacts that look like pores. It is his belief that demonstrable intra-alveolar apertures or communicating ways do not exist normally, although they may be present in pneumonia and other pathological conditions.

On the other hand, certain physiologists and experimental pathologists, notably Van Allen and Lindskog[20] have demonstrated functional if not anatomical intra-alveolar connections or "pores" through which air passes quite readily, and have thus indicated that atelectasis must be lobar in extent when it exists. They assert that there can be no true atelectasis short of that involving an entire lobe unless it be brought about by diseased alveolar walls which act as barriers to the air in neighboring air spaces. If local atelectasis is proved to be nonexistent, this phenomenon cannot explain the local or partially lobar character of the consolidated areas under consideration. Another fact against this contention is this, that physical examination is usually quite different in pneumonic consolidation and massive collapse and is highly characteristic in each condition. In massive collapse the physical examination and roentgen ray reveal retraction of the affected side, elevation of the diaphragm and displacement of the mediastinal structures toward the side of the disease.

As to edema as a cause of the roentgenological appearance under review, it may be said briefly that the very highly developed lymph drainage apparatus of the lungs, with abundant anastomoses in its vast network, the localized nature of many of these consolidated areas, the manner of clearing, the roentgen appearance of known pulmonary edema, and the physical signs all indicate that it has little to do with the formation of these characteristic shadows. Several of these and other possible etiological factors have been well reviewed and considered very recently by Reichle.[15]

Pneumococcus pneumonia occurs in tuberculous patients but it does so only rarely in the patients who live the relatively protected and sheltered life which the sanatorium offers. And the diagnosis is usually not difficult, with the characteristic onset, sputum, bacteriological findings, course and crisis. The pneumococcus may lead to lobar or bronchopneumonia in tuberculous individuals, but in no pneumococcus infection does the illness run the long, protracted course with the relatively few symptoms that characterize the typical consolidation in the tuberculous.

That a peculiar, nonspecific pneumonic consolidation may occur in tuberculous individuals (and in these only) is the now well-known contention of Eliasberg and Neuland,[5] who gave the affection the name "epituberculosis." The only criteria for ordinary pneumonia which these consolidated areas offer are the physical signs of consolidation. On the other hand, it seems clear that they offer several points which make the diagnosis of tuberculosis highly probable if not mandatory. These features are: (1) the long duration, (2) the lack of stormy symptoms, (3) the necessary presence (according to the authors' definition) of tuberculous infection as evidenced by the tuberculin reaction, (4) the presence of tubercle bacilli in the sputum as demonstrated in numerous instances, (5) the histological appearance of excised pulmonary tissue in a clinical case, and (6) the reappearance of the condition in children after injection of tuberculin.

The specificity of this type of pneumonic consolidation cannot seriously be doubted in the face of the last three facts just mentioned, as brief comment upon each will show.

(4) As a rule, tubercle bacilli are wanting in these cases but in one of our patients, cited as illustrative, they were present for a brief period during the height of the consolidation but were absent on repeated sputum examinations before and after this event.

This patient was a child, aged five, whose father had died from tuberculosis two weeks before our first contact with the child. This

contact revealed a marked reaction to the Pirquet test and signs of extensive tuberculosis of the right lung and the region of the root. While a patient in the sanatorium during the next year and a half, he grew normally and experienced marked absorption of the pulmonary infiltration so that there were only a few linear shadows in the region formerly diseased. At this time the tracheobronchial lymph nodes had become distinctly enlarged. Five months later, i.e., two years after detection, while still an inmate, the boy began to feel badly. His temperature rose occasionally to 101° F. and his pulse was accelerated, and in a six weeks' period he lost the 10 pounds he had gained during his

FIG. 1. Roentgenogram of the lung of a five year old boy showing an area of consolidation near the root of the right lung. This process was considerably more extensive five months ago but the film is not available.

stay. At this time he showed physical and roentgenological signs of massive consolidation in the same region of the right lung that had previously been involved. On one occasion during the time when the consolidation was pronounced, the sputum contained tubercle bacilli but it was negative before and has been negative since. The lesion subsided more quickly than is usual in "epituberculosis." The film taken three months later showed marked resorption and the one now (two and a half years later) shows little besides a few linear shadows. The child is in apparent good health. The films

FIG. 2. Roentgenogram of the same boy one year later. There has been marked clearing of the pulmonary lesion, associated with distinct increase in shadows in the mediastinum from enlargement of the tracheobronchial lymph nodes.

illustrating various stages of this child's disease appear as Figures 1 to 5.

(5) An almost compelling bit of evidence for the specificity of the condition has been given by Rubinstein[17] who examined pulmonary tissue removed by biopsy from a

FIG. 3. Film of the same boy taken nearly seven months after that shown in Figure 2. It reveals a marked recrudescence of the disease in the right lung at the former site. This was not present three months before this film was taken. Tubercle bacilli were present in the sputum at this time.

FIG. 4. Film taken approximately three months after that shown in Figure 3. There has been a marked degree of absorption.

consolidated mass that presented all the prerequisites for the diagnosis of "epituberculosis." The tissue proved to be tuberculous as his description and illustrations well show.

Still further evidence of specificity has been furnished by Langer[9] who has related the story of a child, aged four and a half, with an infiltrative lesion in the left lung that gradually resolved during eighteen months of observation. When he was about eight and a half years old, the boy became thin and weak but had no detectable evidence of disease. A Pirquet test was very strongly positive and a subsequent injection of 1/10 mg. of Old Tuberculin led promptly to high fever, symptoms and signs of pulmonary disease which by physical and roentgen examination was found to be consolidation involving the area which had been diseased formerly. This cleared in fourteen days and the child was quite well two years later. It was Langer's contention that tuberculin would not have elicited such a reaction in any save highly allergic, infected tissue. More recently a rather similar case was reported by Goldberg and Gasul.[7] Still further corroborative evidence for tuberculous specificity has been submitted by Ickert[8] by citation of a case of large "infiltrate" on one side, during the resolution of which a similar one, appearing on the other side, proceeded to cavitation and subsequent healing. The case was thought with certainty to be one of tuberculosis. Such data as these are at least highly suggestive of the specific tuberculous character of these affections.

Thus it may be assumed tentatively that such processes are tuberculous. Is the assumption susceptible to proof by experimental methods? Is it possible to reproduce or simulate such lesions in animals? If not, may some of the conspicuous features of these pneumonic, consolidated areas be reproduced under experimental conditions that allow careful analysis? It cannot be stated with certainty that this clinical entity may be reproduced in its entirety but it is quite true that tuberculous pneumonia may be established readily and that the subsequent course—whether of progression or of resolution—is susceptible of careful roentgenological, histological, bacteriological and biological study. Resolution is one of the conspicuous features of pneumonic consolidation and is the feature which has received considerable attention in the study to be reported.

FIG. 5. The film as it appeared twenty months later than the one shown in Figure 4. The boy was clinically well at that time and remains so now after six additional months.

A considerable volume of experimental work has been done on tuberculous pneumonia. In 1905, Nichols,[13] in a beautiful study, inoculated tubercle bacilli into normal rabbits and rabbits that had been infected previously. He noted: (1) that large areas of pneumonic consolidation, which caused the lungs to be very voluminous and noncollapsible, followed from five to nine days after inoculation in the previously infected group; (2) that similar reaction appeared in the other group in the middle of the third week at a time when the lesions in the first group were subsiding, and (3) that in the second group the disease was unremittingly progressive (at least throughout the experiment). The histological appearance of these areas of disease was clearly that of tuberculosis and it was usually pneumonic in character. In 1914 Bezançon and Serbonnes[4] produced specific tuberculous pneumonia in previously infected animals, by intratracheal inoculation of tubercle bacilli. Austrian and Willis[2] gave bovine tubercle bacilli intratracheally to two groups of rabbits. Those in one group were normal and those comprising the other had been inoculated subcutaneously with human tubercle bacilli a few weeks earlier and were, therefore, allergic or sensitive. In the former group the inoculated strain induced ordinary, more or less nodular tuberculosis which appeared grossly in a week or ten days, developed rapidly during the ensuing two weeks to the formation of extensive disease with large areas of consolidation, and eventuated in death. In the sensitive group they produced, twenty-four to forty-eight hours after inoculation, areas of consolidation, gelatinous or chocolate-brown in color, which varied in size from a diameter of a few millimeters to a degree which involved virtually an entire lobe and which, histologically, were composed of a specifically tuberculous reaction. Several of the animals died within ten days of the intratracheal inoculation but several also went through resolution of the pneumonia, as evidenced by serial roentgen examinations, although they died eventually with chronic tuberculosis. Soper, Sampson and Haskins[18] produced pneumonia by intranasal instillation of bovine bacilli in normal and sensitive rabbits. In both groups pneumonia developed but it appeared earlier in the sensitive group. These authors gave very convincing roentgenological proof of resolution of tuberculous pneumonia. They also pointed out the fact that, among workers in tuberculosis, resolution of specific tuberculous pneumonia is a not uncommon observation. This question has received careful study by Gardner,[6] and many characteristics of the process have been described by him. Larson and Long[10] produced pneumonia in guinea pigs by the introduction of tuberculin into the lungs and concluded, as did Rich and McCordock,[16] that the response in the two groups differed greatly in a quantitative but not in a qualitative way. Austrian and Willis[3] reproduced almost the same response in rabbits when they injected tuberculin as when they inoculated tubercle bacilli intratracheally, except that the reaction was less intense. In sensitive animals pneumonia developed frequently after such a procedure, while in normal animals a relatively slight, nonspecific, irritative alveolitis followed the injection.

Such observations as these make plain the fact that hypersensitiveness established by infection with tubercle bacilli is one of the prerequisites to the formation of tuberculous pneumonia—a fact well understood by most workers in tuberculosis and a fact in line with the contention that children having so-called epituberculosis all react to tuberculin.

EXPERIMENTAL DATA

The work herein reported was planned as a study of the course of experimental tuberculous pneumonia especially from the standpoint of specificity and capacity to resolve. The pertinent technical details of the experiment were these: Normal, healthy adult rabbits were used. Those designed for preliminary infection were inoculated

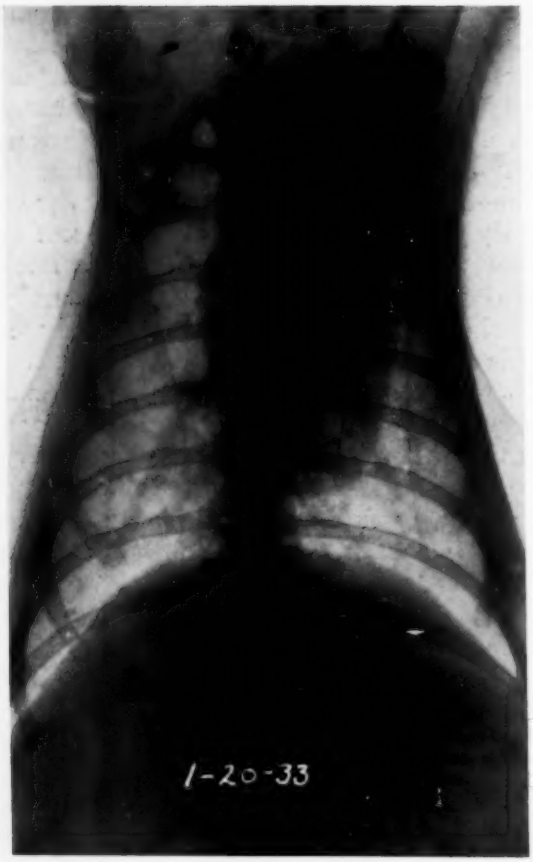


FIG. 6

FIGS. 6 to 9. A series of roentgenograms taken of a rabbit which developed pneumonia following intratracheal inoculation of tubercle bacilli. Several stages of resolution are shown. The film shown in Figure 6 indicates a normal chest. The next day the inoculation was done and Figure 7 indicates a massive pneumonic reaction forty-eight hours later. Eight days after that film was taken, roentgen examination showed beginning resolution (Fig. 8). Finally the film taken forty-three days after inoculation (Fig. 9) showed that the pneumonia had practically cleared. This was confirmed by necropsy.

subcutaneously in the inguinal region each with 1 c.c. of a cloudy suspension of human bacilli (H 37). Approximately one month later animals from this group and also from the group of normals were inoculated intratracheally in equal numbers with suspensions of either human or bovine tubercle bacilli in varying dosage or were injected with tuberculin. These were then studied by frequent roentgen examination and some of them were killed at varying intervals for gross and histological examination of the tissues. Intratracheal inoculation was done by insertion of the needle through shaved skin into the trachea which was held in position by the operator. When short withdrawal of the plunger brought air freely into the syringe, the needle was known to be in proper position, and injection of the suspension was made with the animal on its left side. All animals had preliminary roentgen examination. And all were followed with such examination at frequent intervals after reinoculation. Altogether sixty-two animals were used.

Response to Inoculation with Human Tubercle Bacilli

Previous experience[2] has shown that intratracheal inoculation of bovine tubercle bacilli into normal, noninfected rabbits

FIG. 7

leads to the orderly development of extensive, progressive and fatal tuberculosis (which may be studied by roentgen ray and necropsy) and that similar inoculation of human bacilli produces extensive pulmonary tuberculosis which usually undergoes resolution but which eventuates in death in about one-half of the animals when large doses are employed. Therefore, only a sufficient number of normal controls were used in this experiment to check the viability and pathogenicity of the cultures. By far the larger portion of the experiment was concerned with the introduction of human tubercle bacilli intratracheally into sensitive (previously infected) animals because this setup offered the best basis for resolution of tuberculous pneumonia since the latter could be established by this method at almost every trial. Each received 1.0 c.c. of a milky suspension of

Fig. 8

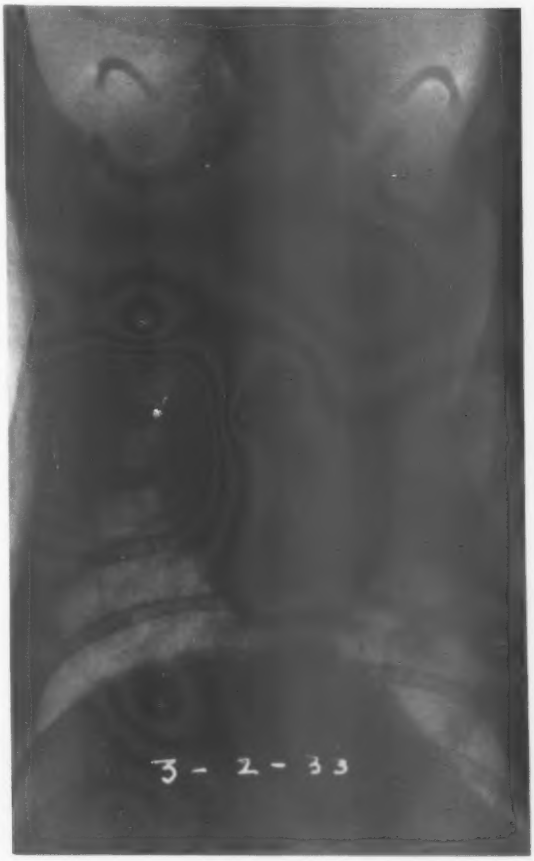


Fig. 9

human bacilli subcutaneously in the right groin. Approximately one month later they were divided into two lots, into one of which 1 c.c. of a heavy, milky suspension of H 37 was inoculated intratracheally and into the other 1 c.c. of a thin, very slightly opalescent suspension of the same strain.

1. *Roentgen Appearance of Experimental Animals.* The day before inoculation in some instances and immediately before in others, roentgen examination was made of the rabbits. This was repeated two days later and thereafter at intervals of a week or ten days. In almost every animal in both allergic groups, shadows of greater or less degree were present in the films. One-half of the animals receiving the large dose died during the first two weeks following inoculation and thus serial roentgenograms revealed no resolution. Several of the survivors and every one in the small dose

group revealed obvious signs of resolution of the shadows in the films; some partial, others complete. This sequence of events is shown in the accompanying series of films (Figs. 6, 7, 8, and 9). Figure 6 represents a clear film that was taken twenty-four hours before the intratracheal inoculation; Figure 7 shows a large, moderately dense shadow involving more than half of the left lung—the film was taken forty-eight hours after inoculation. Figure 8, taken ten days after inoculation, shows a shadow which is much less dense than the one (Fig. 7) taken at forty-eight hours and indicates that considerable absorption had occurred. The final film (Fig. 9), taken forty days after inoculation, shows that the shadows formerly seen had almost entirely disappeared. It may be noted that the consecutive events which are recorded in this series of films compare in many points with those shown in Figures 1 to 4 and compare, except for time relationships, with those seen ordinarily in the case of epituberculosis.

2. *Examination at Autopsy.* Do these shadows represent specific tuberculous pneumonia? May the process not be induced by other bacteria which were introduced by accident? Such bacteria are assumed to be the cause of so-called epituberculosis. Is there unquestioned proof of the specifically tuberculous nature of these reactions? Gross and microscopic examination was made of the diseased tissues of animals sacrificed at stages of the consolidation and shadow formation from early to late both in cases that showed progression and in those that showed regression or resolution.

(*a*) *Macroscopic Examination.* Grossly the average lesion casting a shadow such as that shown in Figure 6 presents itself as a patch of firm tissue of gelatin-like consistence which is semitranslucent, gray or chocolate-brown in color, and which tends to project beyond the surface of the remainder of the organ and thus to stretch the pleura until it is smooth and taut. Toward the fourth, fifth or sixth day a yellow area may appear at the center. This may measure 1 to 2 mm. in diameter or it may involve the larger volume of the consolidation, in cases in which there is progression. The reaction is practically always on the side of the animal which was pendant during the inoculation. Not infrequently, smaller, similar areas of reaction are present on the opposite side also. This disease may progress and lead to death or to resolution and healing. In the former event the area involved is usually extensive at the beginning and it rarely increases very much in size before the animal dies. Frequently it involves an entire lobe or nearly an entire lung. In the course of a week or ten days the diseased area undergoes a transformation from a moist, gelatinous, semitranslucent appearance to that of a dry, opaque, brownish-yellow tissue marked with islands of clearer yellow of varying size which grow larger as caseation proceeds. The diseased lung is usually voluminous and nondistensible. Cross-section view shows that the lung is usually solid throughout most of its depth. In the nonlethal, resolving cases the area, although large, retains its pellucid character throughout but at times it may develop small points of caseation which eventually leave no sign in the healed lung. The process of resolution proceeds at a rate that is more or less comparable to the extent and intensity of the reaction and thus, probably, to the dose. The consolidated area simply becomes smaller with the passage of time. At first the gelatinous tissue involves a considerable portion of a lobe. From ten to twenty-one days after inoculation the area becomes smaller and the pleura over it is somewhat depressed below the pleural level, whereas earlier the diseased area bulged. After another ten days or two weeks the area is still smaller, and tissue which formerly was the seat of extensive infiltration (as shown by serial roentgenograms), assumes a normal appearance. From six to twelve or fifteen weeks after inoculation, the process is one of gradual restitution to normal. Late in

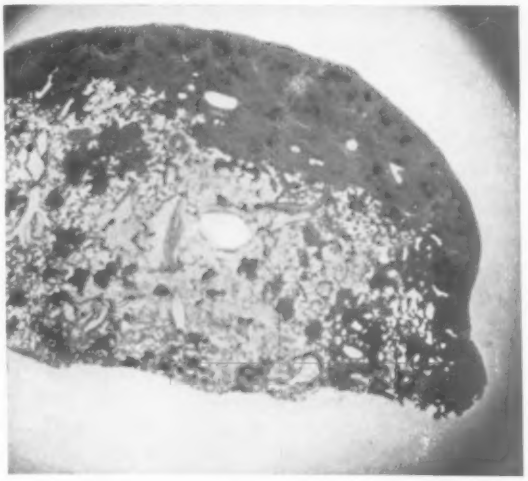


FIG. 10

FIGS. 10 to 13. Photomicrographs illustrating tuberculous pneumonia in varying stages of resolution. Figure 10 represents the reaction to a large dose of tubercle bacilli six days after intratracheal inoculation. The black points indicate caseation. Figure 11 shows all that is left thirty days after inoculation of small dose of tubercle bacilli. Serial roentgenograms indicate that formerly the lobe shown here was almost completely consolidated. Note the depression of the pleura over the area. Figures 12 and 13 represent the appearance four months after a large intratracheal inoculation. The lungs were markedly consolidated and cast a dense shadow earlier in the process. A few small collections of monocytes represent the only residue of the pneumonic reaction.

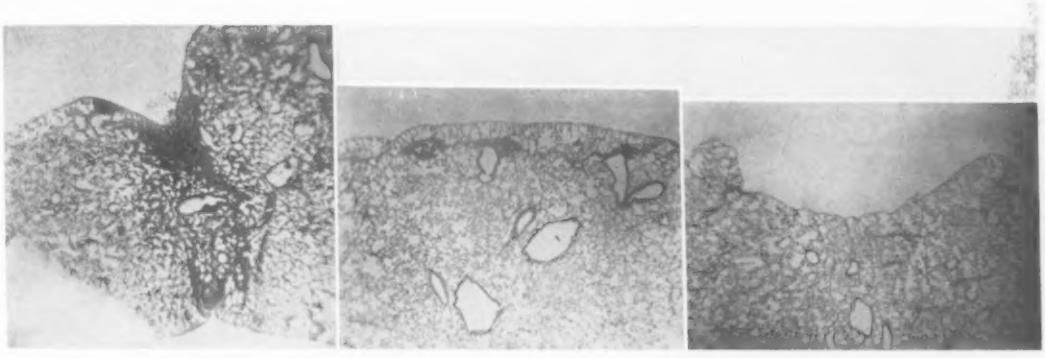


FIG. 11 FIG. 12 FIG. 13

the course of the disease the involved area appears as a small, depressed spot or narrow, depressed groove in the surface of the pleura. It is gelatinous and semitranslucent in appearance and gray-brown in color. Finally, every trace is removed, and the lung appears normal on gross inspection. The cut surface of these lungs at varying stages in the course of the disease reveals the fact that the consolidation is more marked toward the periphery and in midzone although examples are plentiful of consolidation which extends through the whole depth.

(*b*) *Microscopic Examination.* The microscopic appearance of these areas of reaction corresponds to the well-known appearance of exudative tuberculosis. The lungs examined forty-eight hours after intratracheal inoculation reveals consolidation of widely varying degree and extent. The average picture at this stage, casting a shadow such as that shown in Figure 7, is one in which the alveoli are filled with cells and serum. The cells are of three types: first and most abundant are the ordinary epithelioid cells or monocytes, which are rather large and which possess nuclei that are often eccentric in position; secondly, small round cells, and thirdly, polymorphonuclear leucocytes. In places the cells are so numerous as to be packed into the alveoli; in other and more commonly seen areas they are less numerous and fill the alveolus about two-thirds full while frequently serum occupies the remaining space. The predominance of serum over cells which is commonly observed in tuberculous pneumonia

in man is not the rule in experimental animals, although it is frequently observed. In the former, the amount of serum present in the alveoli is conspicuous as compared to the number of cells present. The capillaries are distended, and the alveolar walls—where they can be distinguished from the cellular contents of the spaces—are thickened.

From two to six days the changes are relatively slight but for the appearance of numerous multinucleated giant cells and beginning caseation. The consolidation becomes more compact and larger (often). On the sixth day there are numerous small areas of caseation (Fig. 10) in cases in which large doses were given. Areas toward the periphery of the consolidation contain more serum and fewer cells (Fig. 14). At this stage numerous giant cells of syncytial appearance are present, and the areas of caseation begin to develop in the manner illustrated in Figure 15. In this figure is illustrated a stage in the rather early development of tubercle and is seen characteristically when the focal lesion is from six to twelve or fifteen days old. It shows the nuclear fragmentation and the granular appearance of beginning caseation. In noncaseating areas it illustrates the alveolar exudate in which large, oval monocytes, often with eccentrically placed nucleus and not uncommonly with a pale central zone, are seen. Numerous polymorphonuclear leucocytes are present in and near the caseated regions and a few are scattered elsewhere. Many of these have minute granules that are eosinophilic. The outlines of the alveoli are clearly visible in several places. Caseation becomes very extensive in the animals that die, so that by twelve or fifteen days large areas are apparent, especially toward the central portions of the consolidated tissues. In later stages they appear as large, pale areas that are more or less devoid of nuclear material and demonstrable intact cellular structure.

From the first day of the inoculation, polymorphonuclear leucocytes are present in varying numbers, and especially so during the period of caseation. As time goes on these assume fine eosinophilic granules in increasingly large numbers so that in

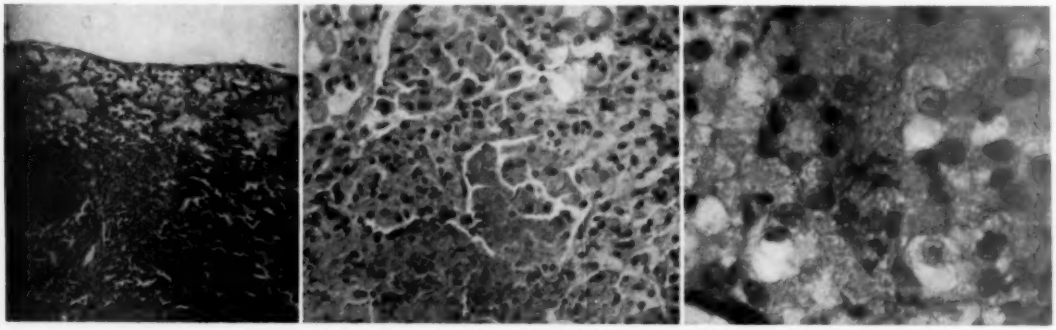


FIG. 14 FIG. 15 FIG. 16

FIG. 14. Photomicrograph of tuberculous pneumonia in a rabbit six days after intratracheal inoculation. Dense cellular and serous or gelatinous consolidation are present (moderately high magnification).

FIG. 15. Photomicrograph of an area of tuberculous pneumonia in which caseation is beginning. The darker portion of the section shows the nuclear fragmentation and cellular stippling characteristic of early caseation. The alveolar outlines are readily determined. The predominating cell is the epithelioid, and several of these have eccentric nuclei and clear central zones (high dry magnification).

FIG. 16. Photomicrograph of an area of tuberculous pneumonia showing a group of tubercle bacilli (oil immersion).

some of the late stages the entire section is heavily speckled with them.

The roentgen-ray and the gross and microscopic examination of the lungs indicate clearly that resolution of massive areas frequently occurs. Is there a characteristic histological appearance to the diseased tissue during this process of resolution? If so, of what does it consist? The histological differences from stage to stage are comparatively scant, aside from the obvious lessening in size of the areas. The area becomes definitely and progressively smaller. The clearing appears to begin in the periphery of the lesion because, in such areas, as time passes there are small patches of partial consolidation lying within a zone in which many alveoli contain a few monocytes and in which there is considerable thickening and cellular infiltration of the alveolar walls. Such cells as remain retain their former relationships: there are just fewer of them. In the more central areas where only a little resolution has occurred, the monocytes and lymphocytes are still present in abundance and the polymorphonuclear leucocytes are fewer in number. As clearing proceeds, the monocytes assume a stippled appearance, which, however, in the highest magnification, is observed to be due to the fact that they have come to possess innumerable, very minute vacuoles. These cells present no further evidence that they have sustained damage. There is no evidence pointing to the fact that the cells in the reaction undergo autolysis or destruction *in situ*.

The process of resolution consists of the disappearance of the cells that cause the consolidation. It is unaccompanied—up to four months, when clearing is often (usually) complete—by the formation of fibrous tissue. Areas of consolidation appear to become less densely consolidated and more patchy, with an increasing number of alveoli containing air, the amount of aeration increasing as the consolidating cells grow fewer in number. What becomes of the cells is not clear. In this experiment the pulmonary tissue previously involved in extensive consolidation presents no trace eventually of the former affect and appears quite as clear as the lung does after lobar pneumonia has completely disappeared. The assumption that the tissue had been the seat of consolidation is based on roentgen study of the animals at frequent intervals between inoculation and sacrifice and on autopsy study at varying stages of the disease.

Examination of tissue stained to show elastic and connective tissue fibers reveals the former in their normal location and quantity in pneumonic areas and shows no abnormal deposits of the latter. Apparently, resolution of this type of tuberculosis is unassociated with the deposition of scar tissue and in this respect is quite unlike chronic fibroid pulmonary phthisis.

1. *Bacteriological Examination of the Tissues.* It is assumed by numerous observers that clinical cases of protracted pulmonary consolidation are brought into being by non-acid-fast bacteria. Careful bacteriological examinations have been made of many sections of tissue in all stages of this experimental disease, both in progression and during resolution. Alternate sections of tissue have been stained for tubercle bacilli and for non-acid-fast bacteria. Many sections have been stained by MacCallum's method[11] which detects both Gram-positive and Gram-negative bacteria in the tissues, but no such bacteria have ever been found. Tubercle bacilli have been detected in the pneumonic tissue at almost every stage of the process. In some instances they may be found readily; in others prolonged search is required. They may lie singly or in small clumps and may be free in the tissues but are much more likely to be found lying within giant cells or epithelioid cells. In Figure 16 a photomicrograph reveals a small clump of tubercle bacilli which lie within an area of consolidation that is composed almost solely of mononuclear cells.

Response to Tuberculin in Normal and Allergic Animals

Into each of 9 normal rabbits, 1 c.c. of a 2.5 per cent solution of Old Tuberculin

was injected and into 7 normals 1 to 2 c.c. of a 2 to 50 per cent solution of T. P. T.* was so injected. Of these 16, killed from one day to one week after injection, 3 showed in the gross a definite hemorrhagic alveolitis composed chiefly of small round cells and red blood cells; in all there was congestion, dilatation of capillaries and extrusion of red blood cells in greater or lesser numbers, and in one there was a low

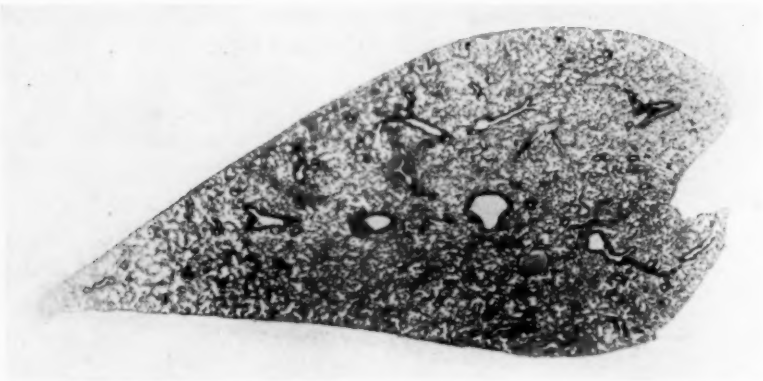


FIG. 17. Photomicrograph—very low power—of a section from the lung of a normal animal killed two days after injection of tuberculin intratracheally. The only reaction is a mild alveolitis.

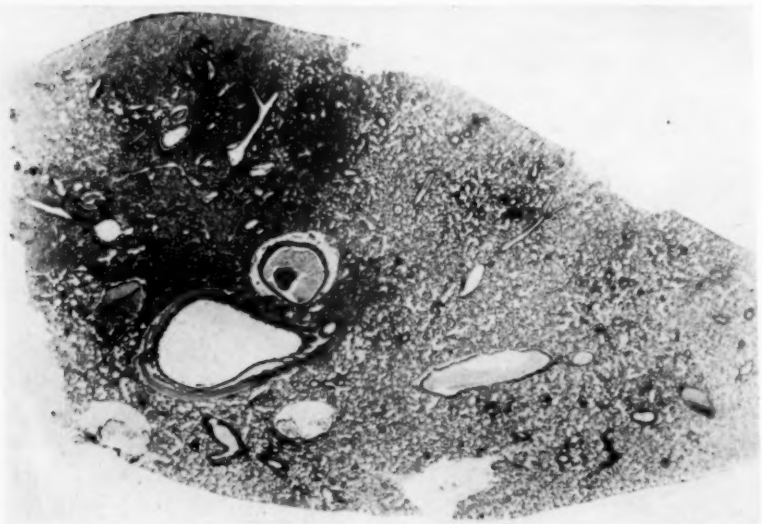


FIG. 18. Photomicrograph showing the forty-eight hour response of an allergic animal to the injection of tuberculin. A "tuberculin pneumonia" has been established (cf. Fig. 17).

discoloration of the lungs which, however, were soft and not consolidated. Histologically these and almost all of the others showed a moderate degree of nonspecific grade, patchy pneumonia composed almost exclusively of small round cells. This animal was killed seven days after injection. Figure 17 illustrates the histological appearance seen forty-eight hours after injection of tuberculin in the average non-

* T. P. T. is a purified protein obtained from the tubercle bacillus by Seibert's method. It was kindly furnished by Parke, Davis and Company.

sensitive animal (cf. Fig. 18). Roentgenograms were taken of all and showed nothing.

When tuberculin was injected into the trachea of *16 previously infected animals*, acute pneumonic reaction occurred in 7 as shown by roentgen examination and in 12 as shown grossly at autopsy. Some of these had had one and some several injections of tuberculin. In none of the animals was the process of resolution followed roentgenologically, but it has been shown by Larson and Long[10] that resolution occurs promptly and thus that the pneumonia is relatively evanescent. The microscopic appearance was quite like that which followed inoculation of tubercle bacilli, as shown in Figure 18.

DISCUSSION

Objection may be raised to this work because of the fact that clearing of the ensuing disease is usual when human tubercle bacilli are inoculated into rabbits. However, four facts validate the deductions that may be made from the work. These are: (1) human tubercle bacilli in sufficiently large dose leads to death in one-half the animals inoculated; (2) reinfection of rabbits with bovine bacilli produces tuberculous pneumonia which frequently follows the early course described for human bacilli, in the animals in which the response is not promptly fatal. In most, if not in all, rabbits infected with bovine tubercle bacilli death is the eventual result, but in 30 per cent of those which have acute tuberculous pneumonia there is a marked degree of resolution of this process before the generalized disease becomes chronic and unremittingly progressive; (3) the fundamental pathological response appears to be similar in both instances; (4) demonstrated resolution of tuberculous pneumonia induced by bovine bacilli has been clearly shown.[2,18]

Is it permissible to conclude that the pneumonic consolidation in these experimental animals is specifically a tuberculous process and that it arises without relation to other bacteria? The facts justify this conclusion when it is recalled (1) that the reaction occurs only in animals with previous tuberculous infection—i.e., in specifically allergic animals; (2) that inoculation of tubercle bacilli in pure culture induces the reaction in such animals but not in nonallergic animals until they have become sensitive through the infection; (3) that intratracheal injection of nonspecific substances such as blood, salt solution and suspensions of carbon particles does not produce such a response; (4) that other bacteria so introduced initiate abscess or chronic pneumonia but not this type of response;[21] (5) that tubercle bacilli are demonstrable in the consolidated tissue (as they are also in the tissues of clinical tuberculous pneumonia) and no other bacteria can be found; (6) that tuberculin will bring about this same type of specific reaction when introduced intratracheally into sensitive (infected) animals.

Is it permissible to deduce that such a specific response in experimental animals is comparable in principle to the chronic pulmonary consolidation seen clinically in tuberculous people (or in those with tuberculous infection)? Again the facts seem to warrant such a deduction because of (1) the presence of tuberculous infection in such patients—as shown by the tuberculin test—and thus of specific sensitiveness on which the allergic reaction rests; (2) the fact that the administration of tuberculin had been associated with an exacerbation of so-called epituberculosis in children;[7,9] (3) the relative chronicity of the process of resolution; (4) the comparable histological appearance of biopsy tissues of a clinical case, with that of these animals; (5) the comparable histological structure of gelatinous pneumonia in people, with this reaction in animals, and (6) the results of the occasional necropsy made in such clinical cases.

It should be remembered that, clinically, such reactions are by no means confined to children. As a matter of fact, the underlying principles here discussed are doubt-

less at work at times in every case of tuberculosis:—where the early exudative lesion appears in its characteristic seat in the first or second interspace, when the patient has a "spill" into the contralateral lung which leads to an area of fresh disease or whenever a pulmonary exacerbation occurs. All such areas of disease represent examples of tuberculous pneumonia of limited anatomical extent.

CONCLUSIONS

Pulmonary consolidation which occurs with more than occasional frequency in tuberculous individuals is believed by different authors to be due to numerous causes among which are: atelectasis, edema, pneumonococcus pneumonia, nonspecific pneumonia and tuberculous pneumonia.

Tuberculous pneumonia may be induced in experimental animals. This disease frequently undergoes resolution as evidenced by serial roentgenograms and by necropsy. It is clear that this pneumonia in animals is specifically tuberculous in nature.

It is likely that such specific pneumonic processes are very similar to or identical in nature with clinical pneumonic consolidation which occurs in tuberculous individuals, and that each is the product of the exudative, allergic reaction of an animal organism which is sensitive to tubercle bacilli and tuberculin.

At certain stages of this work Dr. J. M. Appel and Mr. Joseph Bobbio rendered very material help, for which I wish to express thanks.

REFERENCES

1. Assmann, H. Frühinfiltrat. *Ergebn. d. ges. Tuberk.*, 1930, *1*, 115–194.
2. Austrian, C. R., and Willis, H. S. The pulmonary effects of intratracheal injections of tubercle bacilli and blood in rabbits. *Am. Rev. Tuberc.*, 1926, *14*, 306–315.
3. Austrian, C. R., and Willis, H. S. The pulmonary effects of intratracheal injections of old tuberculin in rabbits. *Am. Rev. Tuberc.*, 1931, *23*, 310–317.
4. Bezançon, F., and Serbonnes, H. Superinfection tuberculeuse expérimentale du cobaye; étude des phénomènes allergiques cutane et pulmonaire. *Ann. de méd.*, 1914, *1*, 129.
5. Eliasberg, H., and Neuland, W. Die epituberkulöse Infiltration der Lunge bei tuberculösen Säulingen und Kindern. *Jahrb. f. Kinderh.*, 1920, *43*, 88–97. Also: Zur Klinik der epituberkulösen und gelatinösen Infiltration der kindlichen Lunge. *Jahrb. f. Kinderh.*, 1921, *44*, 102–118.
6. Gardner, L. U. Healing by resolution in experimental tuberculosis. *Am. Rev. Tuberc.*, 1922, *6*, 163.
7. Goldberg, B., and Gasul, B. M. Epituberculosis; a study of ten cases over a period of two years. *Am. J. M. Sc.*, 1930, *180*, 824–832.
8. Ickert, F. Spontanheilung einer grossen Infiltrateinschmelzung? *Beitr. z. Klin. d. Tuberk.*, 1929, *72*, 317–323.
9. Langer, H. Die Spezifität rückbildungsfähiger Lungeninfiltrate bei kindlicher Tuberkulose (sog. epituberkulöse Infiltration). *Ztschr. f. Kinderh.*, 1923, *34*, 142.
10. Larsen, A., and Long, E. R. Experimental tuberculin pneumonia. *Am. Rev. Tuberc.* 1931, *23*, 41–44.
11. MacCallum, W. G. A stain for influenza bacilli in tissues. *J. Am. M. Ass.*, 1919, *72*, 193.
12. Miller, W. S. The alveolar pores of pneumonia. *J. Exper. Med.*, 1925, *42*, 779–783. Also: Factors underlying formation of alveolar pores in pneumonia. *J. Exper. Med.*, 1923, *38*, 707.
13. Nichols, J. L. Studies on immunity to tuberculosis; an histological study of the lesions of immunized rabbits. *Med. News*, 1905, *87*, 638–641.
14. Pagel, W., and Henke, I. Lungentuberkulöse. In: Handb. d. speziellen Path. Anat. u. Hist., Henke and Lubarsch, Springer, Berlin, 1930, III, Pt. 2, 139 (esp. 244–248).
15. Reichle, H. S. Resolving exudates in pulmonary tuberculosis of childhood. *Am. J. Dis. Child.*, 1933, *45*, 307–330.
16. Rich, A. R., and McCordock, H. A. An enquiry concerning the rôle of allergy, immunity and other factors of importance in the pathogenesis of human tuberculosis. *Johns Hopkins Hosp. Bull.*, 1929, *44*, 273–422.
17. Rubinstein, H. Zur pathologischen Anatomie der Lungeninfiltrierungen. *Beitr. z. Klin. d. Tuberk.*, 1928, *70*, 773–783.
18. Soper, W. B., Sampson, H. L., and Haskins, C. H. Aspiration tuberculosis in rabbits. *Am. Rev. Tuberc.*, 1927, *15*, 88–110.
19. Stewart, C. A. Pulmonary diseases; 5,816 cases in children, with special reference to childhood type of pulmonary tuberculosis. *Am. J. Dis. Child.*, 1932, *43*, 803–827.
20. Van Allen, C. M., and Lindskog, G. E. Collateral respiration in the lung. *Surg., Gynec. & Obst.*, 1931, *53*, 16–21.
21. Wollstein, M., and Meltzer, S. J. Experimental bronchopneumonia by intrabronchial insufflation. *J. Exper. Med.*, 1912; *16*, 126; also: *J. Exper. Med.*, 1913, *18*, 548.

Vol. XXXI, No. 6

SOME MECHANICAL FACTORS OF GASTRIC PHYSIOLOGY*

STUDY I. THE EMPTY STOMACH AND ITS VARIOUS WAYS OF FILLING. THE PRESSURE EXERTED BY THE GASTRIC WALLS ON THE GASTRIC CONTENT. THE PHYSICAL CHANGES OCCURRING TO THE FOODSTUFF DURING DIGESTION

By CESARE GIANTURCO, M.D.
Fellow in Radiology, The Mayo Foundation
ROCHESTER, MINNESOTA

INTRODUCTION

IN SPITE of numerous experimental and clinical studies on the mechanics of gastric activity, several problems remain which are not completely solved. Some of these problems are of particular interest to the roentgenologist because they concern phases of gastric activity involved in diagnostic roentgen examination of the stomach. I have attempted to study the more important events that occur from the time of entrance of food into the empty stomach until its exit through the pylorus, focusing attention particularly on those phases of the gastric mechanism which are of roentgenologic significance.

Five different studies were made, three of which are reported in this paper; the other two, elsewhere.[3]

As many other observers have done, I used cats as experimental animals. These animals are easily trained, and their stomachs appear strikingly similar to the stomach of man. The methods of investigation were such as to avoid interference with the normal motility of the gastrointestinal tract. Roentgen rays offer the best means of studying the movements of the stomach and duodenum without the use of anesthetics and without opening the abdomen. Most of the experiments were carried out by means of roentgen rays. Air or barium suspended in various fluids, or barium-coated solid fragments of food, were used as contrast media. In order to observe the movements of the visceral walls themselves small chilled lead shot were placed under the serosal coat of the stomach. A month after the healing of the operative wounds the animals were trained to lie quietly on a holder; they were observed roentgenoscopically and records were taken in the form of roentgen cinematographic films. These films were taken with the machine devised by Alvarez.[4]

Pressure conditions in the stomach were measured by filling the organ with air through a small tube connected with a syringe and a manometer. By turning a three-way stopcock to different positions it was possible to inject different amounts of air and to measure the pressure obtained at each injection.

THE EMPTY STOMACH AND ITS VARIOUS WAYS OF FILLING

The hypothesis that food which enters the stomach follows the longitudinal mucosal folds (Magenstrasse) along the lesser curvature has gained practical importance since Aschoff and his coworkers have attempted to explain the more common occurrence of ulcer along the lesser curvature by the fact that this region is more traumatized than is the remainder of the stomach. This hypothesis has not generally been accepted. Although some authors reported that in cases of poisoning caused by caustic agents the lesions were localized at the lesser curvature and the remainder of the stomach was intact, other investigators found by feeding animals colored

* Abstract of thesis submitted to the faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Radiology. Work done in the Institute of Experimental Medicine, The Mayo Clinic, under the direction of F. C. Mann, W. C. Alvarez and B. R. Kirklin.

fluids that the color spread all over the gastric mucosa. When the roentgen ray came into use, several investigators worked on this problem with the new method, but the conclusions of the early investigators must be looked on as doubtful because, although they could follow the descent of the opaque food, they could not be sure where the greater and lesser curvatures were when the stomach was empty.

An attempt to visualize the lesser curvature of the stomach was made by Lehmann, who used a duodenal tube opaque to roentgen rays. The tube was swallowed by the subject and, as soon as the distal end was seen in the duodenum, gentle tension was made at the oral end so as to bring the tube in contact with the lesser curvature. Then the subject swallowed some barium. Lehmann concluded from this experiment that the Magenstrasse was not the actual path followed by the food. Later his method was strongly criticized by Lossen and Schneider because the tension on the tube could have disturbed the whole mechanism of the Magenstrasse.

In order to make the Magenstrasse visible roentgenologically, Lossen and Schneider used silver beads sewn to the two curvatures of the stomachs of dogs. These investigators then gave opaque material to the dogs and after roentgenoscopy concluded that the Magenstrasse was the path actually followed by the ingesta.

Because of the technique employed, the work of Lossen and Schneider deserves serious consideration. It is my impression, however, that their conclusions cannot safely be accepted because the beads used in the experiment were poorly placed, and from my experience with their technique I feel sure that they must have produced adhesions about the stomach. As Lossen and Schneider pointed out, it is only when the stomach is empty and relaxed or when it is full of food, that one can talk of a Magenstrasse, because when the stomach is empty and contracted there is only one possible channel for food and that is down the central lumen. This lumen would be in the middle, and therefore a bolus entering it would come in contact with both the lesser and the greater curvatures at the same time.

Before attacking the problem of the Magenstrasse itself, I thought it would be of interest to investigate the conditions of the empty stomach.

Does Peristalsis Occur in the Really Empty Stomach? A tremendous amount of literature has grown up about the phenomenon of "hunger contractions" in the fasting stomach. To me, the question has always remained whether or not these contractions were originally present or stimulated by the recording balloon which has invariably been used. It is well known that distention of a hollow organ such as the stomach commonly gives rise to rhythmic contractions. In order to throw light on this problem, I first placed chilled lead shot along the lesser and greater curvatures of the stomachs of nine cats; after the animals had recovered, they were fasted for two days and then observed for hours with the roentgenoscope. In more than eighty experiments I could detect movement of the gastric walls in only one animal, and the stomach of that one contained a considerable amount of swallowed air.

My impression from this work was that the so-called hunger contractions were caused by distention of the stomach by the recording balloon, but as there was some doubt that my method of visualization would indicate contractions in the empty stomach even if they were present, I took another group of four cats and placed many more shot on the lesser and greater curvatures, so that it was easier to follow the movement of a peristaltic wave, if such should appear. After these animals had recovered from the operation and had been fasted for two days, eight films, each including 100 roentgenograms taken one each second, were made and carefully studied (Fig. 1). In seven of these films, shallow peristaltic contractions could be made out. It appears then that, just as in the bowel, although visual observation may not dis-

close rhythmic activity, graphic records will show that some motion is taking place. It seems probable, however, that the large amplitude of the contractions observed with the usual technique in which a balloon is used is due to the stimulus of distention.

The Behavior of the First Bolus Entering the Empty Stomach. After a fast of two days, the four animals were given 0.5 c.c. of a suspension of barium in water. As soon as this reached the stomach it was carried toward the pylorus by peristaltic waves without following the Magenstrasse (Fig. 2, *a*). Similar behavior was noted when a larger amount of fluid (Fig. 2, *b*) and when solid food was ingested.

The Behavior of Boluses Entering a Full Stomach. The four fasting cats were given 25 c.c. of ground meat, which was followed by 1 c.c. of an aqueous suspension of barium. This small amount of opaque material followed the Magenstrasse, but when 15 c.c. more of barium water was given this could be seen to spread out from the lesser curvature and surround the solid food on all sides (Fig. 3, *a*).

The cats were again fasted; then they were given 5 gm. of meat and next 30 c.c. of barium water. The opaque material immediately surrounded the meat on all sides (Fig. 3, *b*).

Conclusions. 1. The empty stomach is animated by shallow peristaltic contractions or is tonically contracted; under these conditions the Magenstrasse is not functionally distinguished from the remainder of the stomach. This is demonstrated by the behavior of opaque material entering an empty stomach, the walls of which are outlined roentgenographically by small lead shot.

2. The Magenstrasse is followed only when small amounts of material enter a stomach which is already filled by solid food.

3. Large amounts of water or food entering a full stomach do not follow the Magenstrasse, but spread from the cardia out around the food already present.

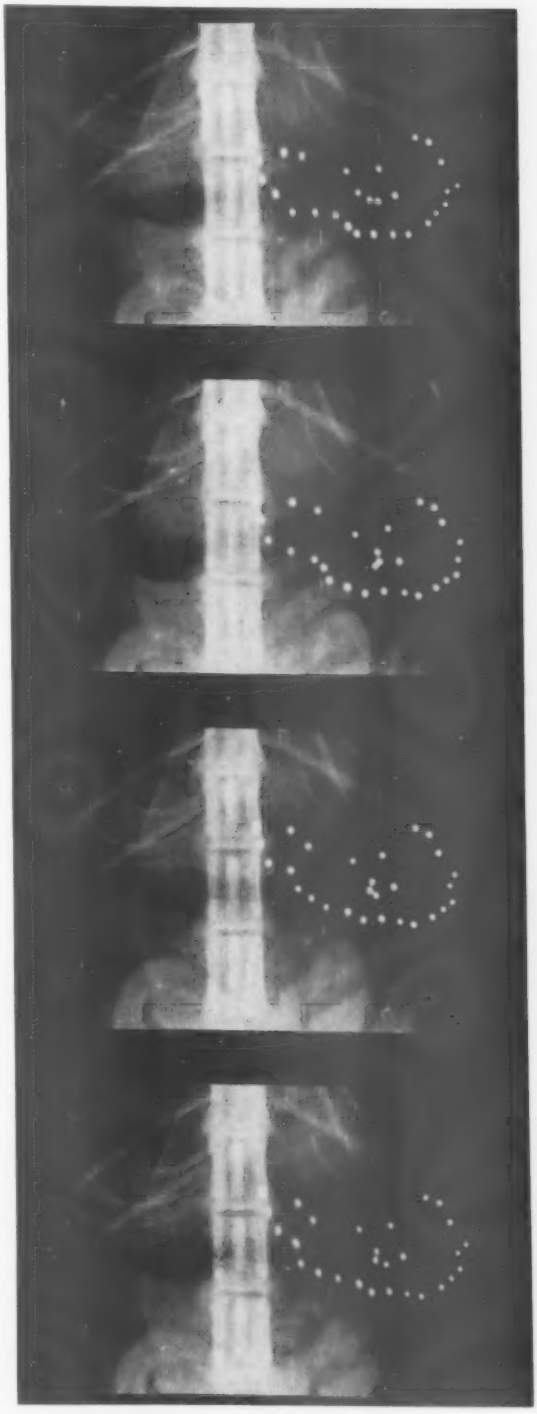
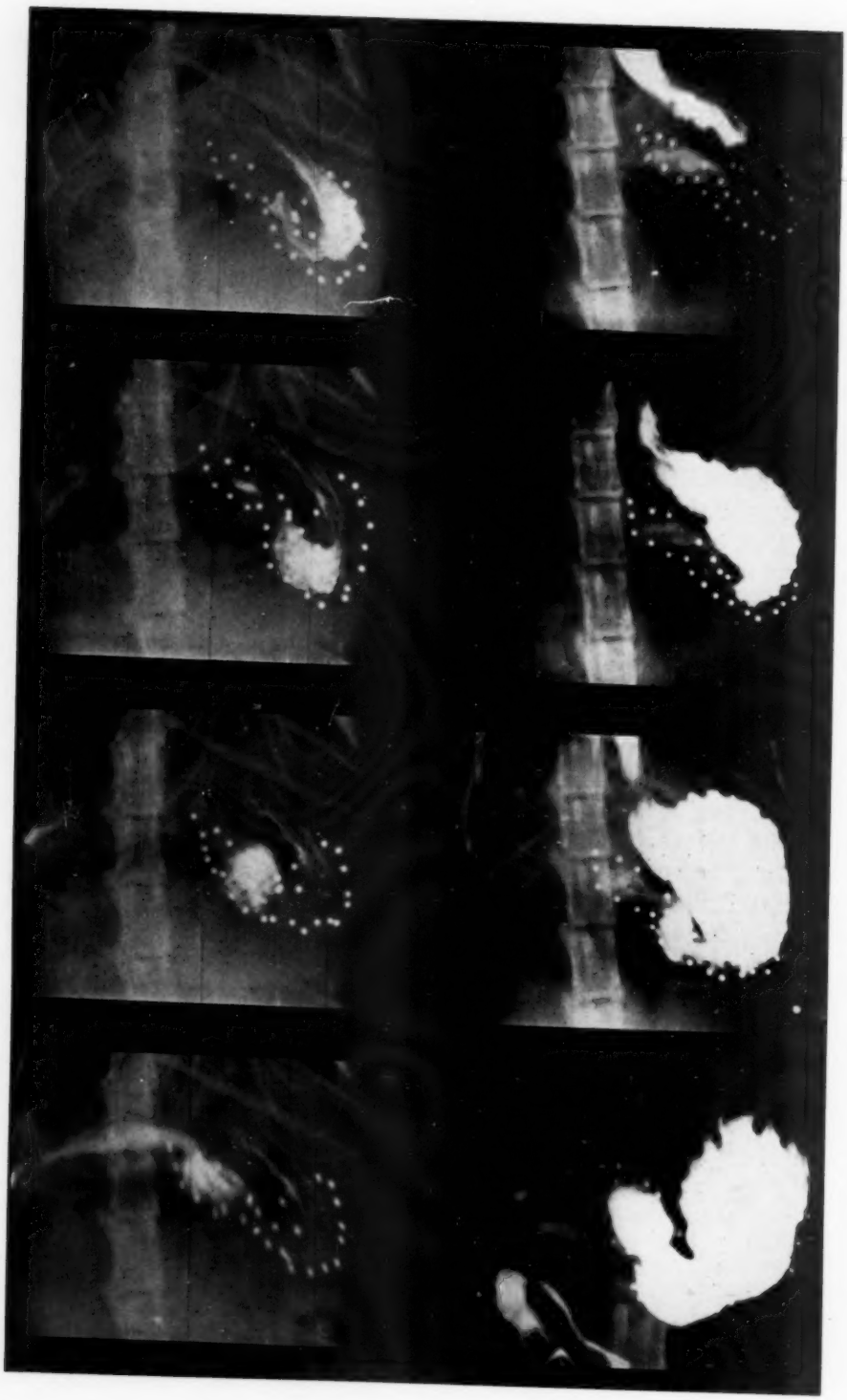
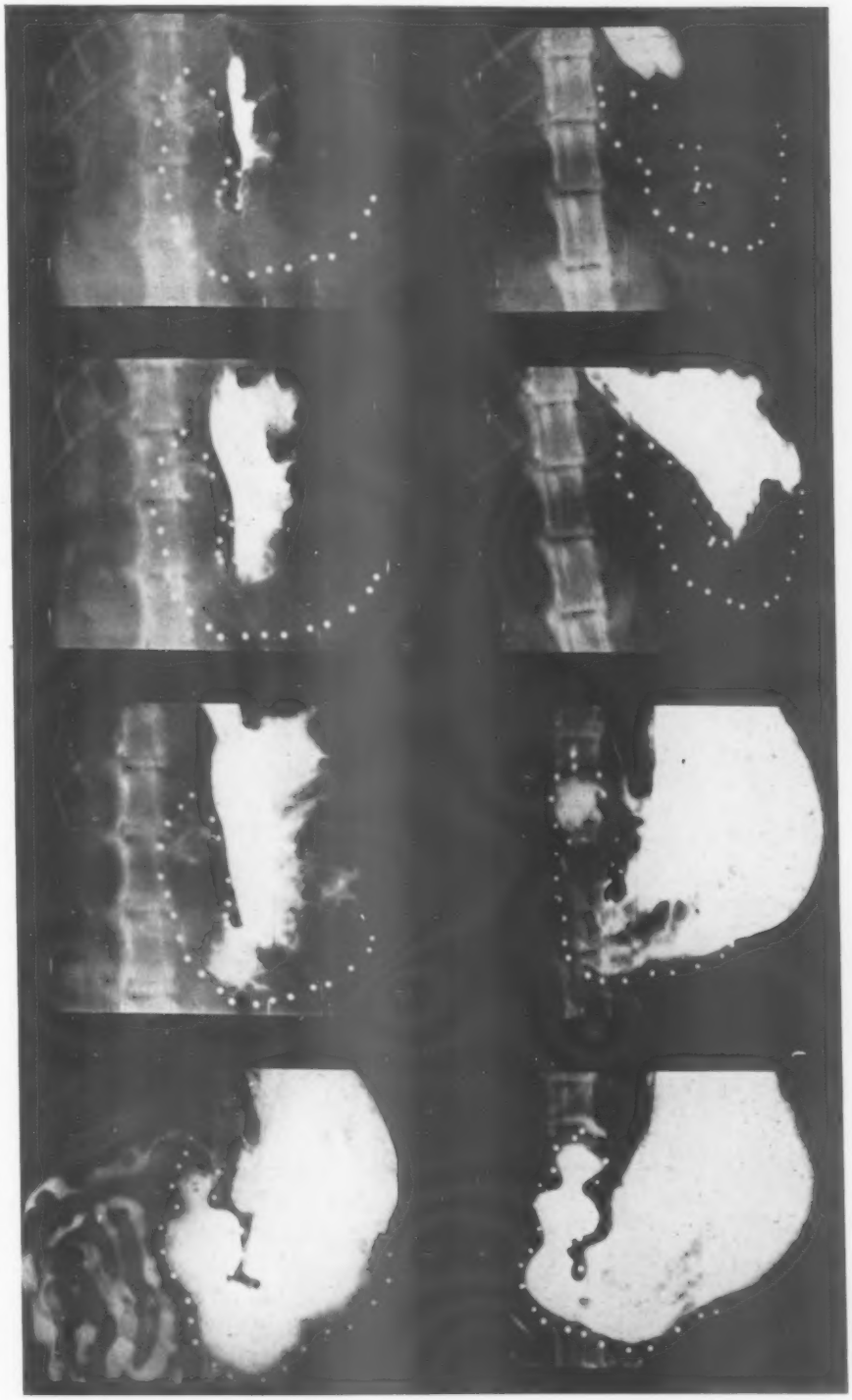


Fig. 1. Peristalsis in the empty stomach shown by the lead shot imbedded in the gastric walls.



a *b*

FIG. 2. Filling of the stomach.



a *b*

Fig. 3. Behavior of boluses entering a full stomach.

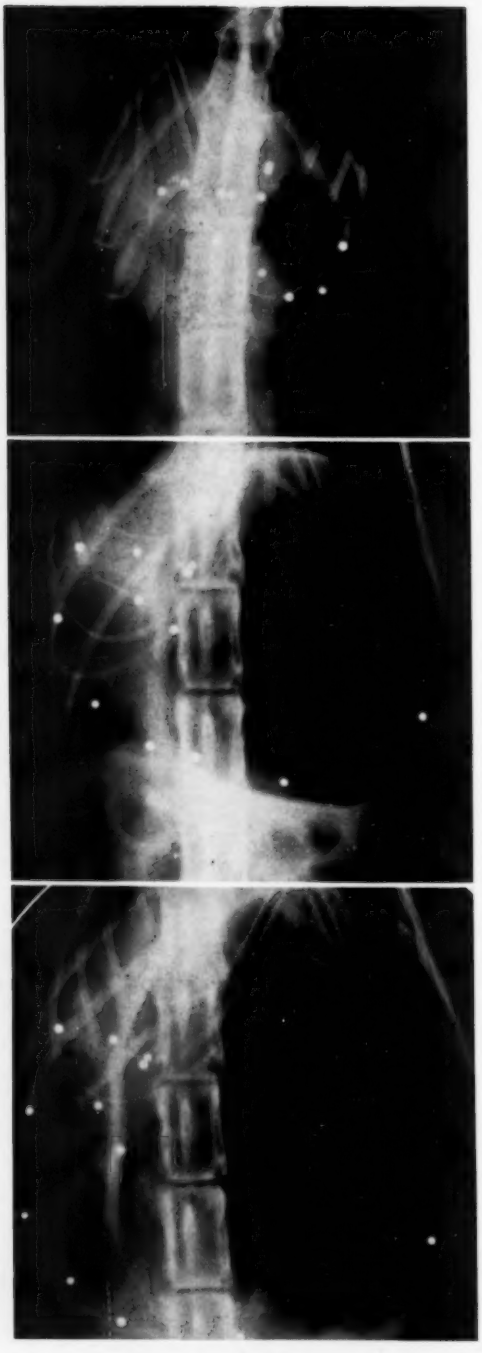


Fig. 4. Relaxation of the gastric walls at different fillings: The top roentgenogram was made after injection of 5 c.c. of air; the middle one after injection of 75 c.c., and the bottom, after injection of 200 c.c.

THE PRESSURE EXERTED BY THE GASTRIC WALLS

Intragastric Pressure during the Filling of the Stomach. The amount of literature on this subject is not large, but the papers published by Kelling and by Moritz at the end of the nineteenth century represent a mine of information. One of the most interesting points brought out by these authors is that the stomach relaxes as it fills, so that up to a certain point intragastric pressure does not increase with increasing volume of gastric content.

I repeated, on four cats, the work done by Kelling and Moritz on human beings and on dogs. The measurements confirmed the conclusions of Kelling and Moritz. With the gastric content increasing from 5 to 100 c.c. there was no increase in gastric pressure, and a peculiar relaxation of the walls of the stomach must be postulated. From 100 c.c. onward this relaxation did not keep pace with the increased volume of the gastric content and the pressure rose rapidly.

The Site of Relaxation in the Gastric Walls. The next question was: Does this relaxation of the wall take place equally all over the stomach, or is it more marked in one part than in another?

In one of the first experiments, the stomachs of two cats, with the gastric walls outlined by means of lead shot, were distended with air. This was injected 25 c.c. at a time and a roentgenogram was made after each injection. By measuring the distances between the shot at the different stages of filling, it was shown that the gastric walls gave way only slightly in the pars pylorica, whereas they stretched markedly in the fundus and in the upper and outer parts of the body (Fig. 4).

I then excised the fundus and the upper and outer parts of the body of the stomachs of two animals, and about a month later, the pressure was measured, using the same technique. Figure 5 shows that in these animals the pressure increased rapidly as the stomach filled. Apparently,

then, those two parts of the stomach are most important in the regulation of intragastric pressure.

Why in the Intact Stomach Does the Gastric Pressure Rise Rapidly when the Gastric Content Reaches a Certain Volume? In attempting to answer this, two possibilities had to be taken into consideration: (1) that there is a limit beyond which the gastric walls do not relax, and (2) that with the increasing volume of the stomach, there comes a point at which the abdominal walls begin to stretch, and abdominal pressure must increase.

In a first experiment the intragastric pressure was measured at the same time as the abdominal pressure, with the animal under ether anesthesia. A tube was placed in the stomach of a cat and connected with the apparatus already described (syringe and manometer). The abdomen was opened in the lower half of the median line; a rubber balloon connected with a second water manometer was placed in the peritoneal cavity and the wound was closed. By means of a syringe, 25 c.c. of air was injected into the stomach about every minute. At the beginning of the experiment the pressure in the abdominal cavity was approximately zero, varying slightly with respiration, and the pressure in the stomach, 4 cm. of water. After 125 c.c. had been injected, however, the manometers began to register a simultaneous increase in pressure in the stomach and in the abdomen. When 300 c.c. had been injected, the pressures were 20 cm. of water in the stomach and 13 cm. in the abdominal cavity. After taking these readings the air was allowed to escape from the stomach.

In the next experiment the abdomen was left wide open and air was pumped into the stomach, 25 c.c. at a time. Under these conditions the gastric pressure began to rise only after 200 c.c. had been injected, and when 300 c.c. had been injected, it reached only 10 cm.

In another cat the order of these experiments was reversed, and the results corresponded closely with those previously obtained. I believe, therefore, that the increase in gastric pressure, which comes after the stomach has been distended to a certain point, is due largely to the force required to stretch the abdominal wall.

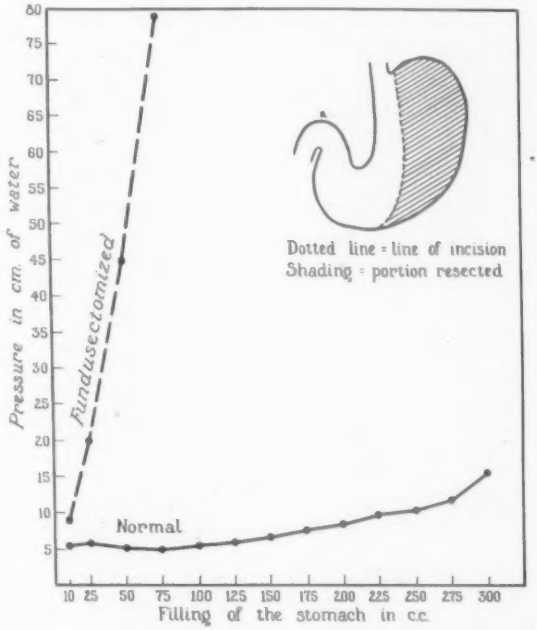


FIG. 5. The parts of the stomach that were excised, and the pressure within the normal stomach compared with that in the stomach of which the fundus had been excised.

The Effect of High Gastric Pressures on the Emptying of the Stomach. The fact that after removal of the fundus it is possible to reach high gastric pressures enabled me to study the effect of high pressures on the rate of emptying of the stomach.

Two cats from which the fundus of the stomach had been resected were given varying amounts of aqueous suspensions of barium (30 per cent), and the emptying time was observed with the roentgenoscope. At the same time two normal cats were observed as controls. Table 1 shows that, compared with a normal organ, the emptying time of a stomach from which the fundus was resected is shorter, the greater the filling and the higher the pressure reached.

Conclusions. 1. Because of active relax-

ation of the fundus and body of the stomach, the intragastric pressure does not change until a large amount of material has been introduced. The rise which follows further filling is largely the result of stretching of the abdominal walls.

2. After the fundus is resected the emptying time of the stomach is shortened.

PHYSICAL CHANGES OCCURRING TO THE FOODSTUFF DURING DIGESTION

Liquefaction of Solid Food. If an animal is killed and the gastric content is observed some time after the animal has eaten solid food, it is found that this food has changed to fluid. Study of this phenomenon is best carried on by direct observation of the sectioned frozen stomach, but by the roentgenologic method one is able to study the process of liquefaction during the whole course of gastric digestion without killing the animal.

TABLE I

EFFECT OF HIGH INTRAGASTRIC PRESSURE ON THE EMPTYING TIME OF THE STOMACH

Water introduced, c.c.	Initial intragastric pressure, cm. of water (fundus removed)*	Emptying time, minutes	
		Normal stomach	Fundus removed
10	9	30	30
25	20	65	40
50	45	110	50
75	79	140	65

* The pressure within the normal stomach was 4 or 5 cm. of water in all instances.

Each of three cats was given a meal of fairly large pieces of meat (each piece about 4 gm. in weight), the surface of which had been coated with powdered barium. The cat usually chews very little, and with the roentgenoscope I could see in the stomach the transparent pieces of meat sharply defined one from the other by the covering of barium. Peristalsis was shallow for the first few minutes, but soon the waves grew deeper and deeper, especially in the pyloric region. About ten minutes after feeding, the sharp lines of barium in the antrum began to fade, and in about twenty minutes the whole pyloric region became diffusely opaque (Fig. 6, *a*, top). At this point one of the animals was killed, and section of the stomach showed that in the pars pylorica the contents had actually become liquefied, and that the barium was finely mixed with the ground meat. One hour after ingestion of the meal the zone of liquefaction had spread back into the body of the stomach (Fig. 6, *a*, middle). Two hours later it was possible to see the sharp contours of lumps of meat only in the central part of the fundus (Fig. 6, *a*, bottom).

The roentgenograms show, as one would expect, that the changing to fluid is a surface phenomenon by which only the most peripheral parts are affected. The early liquefaction of the material in the antrum can be explained by the fact that peristalsis is particularly strong in this region, which well deserves its name, the "gastric mill."

Separation of Fluids from Solid Food. The fact that at the pyloric end of the stomach, food is fluid at an early period of digestion is of the greatest importance in determining the mode of emptying of this organ. As has been shown, the presence of fluid food in the antrum is due to more active peristalsis which induces a local change to fluid; it is due also to the trickling down of fluids from the upper parts of the organ.

Each of three cats was given 20 c.c. of meat cut in large pieces; this feeding was immediately followed by 20 c.c. of milk made opaque by the addition of barium. The first picture, taken soon after the ingestion of milk, showed the negative shadows cast by the transparent lumps of meat lying in the surrounding opaque fluid (Fig. 6, *b*, top). The subsequent pictures (Fig. 6, *b*, middle and bottom) were taken each ten minutes after the one preceding, and show that milk leaves the stomach before meat, which is still to be seen in the organ when almost all of the milk is in the intestine.

a *b*

Fig. 6. *a*, Fluids leaving the stomach before solid food. *b*, Liquefaction of solid food.

I think, also, that the separation of fluids from solid food can be related to the peristaltic activity of the organ, because the waves act much as does the hand of a milker; running continuously toward the pylorus they massage the peripheral part of the solid content and inundate it with gastric juice. At the same time the waves push onward the fluids which result from digestion and those which are ingested. By this remarkable mechanism the fluid part of the gastric content is always kept well down by the pylorus where it is ready to pass into the intestine.

REFERENCES

1. ASCHOFF, LUDWIG. Über den Engpass des Magens (Isthmus ventriculi). Fischer, Jena, 1918.
2. ASCHOFF, LUDWIG. Lectures on Pathology. P. B. Hoeber, New York, 1924, 365 pp.
3. GIANTURCO, CESARE. Some mechanical factors of gastric physiology. Study II. The pyloric mechanism. The effect of various foods on the emptying of the stomach. AM. J. ROENTGENOL. & RAD. THERAPY, June, 1934, *31*.
4. GIANTURCO, CESARE, and ALVAREZ, W. C. Roentgen ray motion pictures of the stomach. *Proc. Staff Meetings of Mayo Clinic*, 1932, *7*, 669–671.
5. KELLING, GEORG. Untersuchungen über die Spannungszustände der Bauchwand, der Magen und Darmwand. *Ztschr. f. Biol.*, 1903, *44*, 161–258.
6. LEHMANN, J. C. Beiträge zur Frage der Magenstrasse. *Arch. f. klin. Chir.*, 1923, *127*, 357–375.
7. LOSSEN, HEINZ, and SCHNEIDER, ERICH. Röntgenstudien über die Magenstrasse. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1926, *34*, 252–261.
8. MORITZ. Studien über die motorische Thätigkeit des Magens. I. Mittheilung. Über das Verhalten des Druckes im Magen. *Ztschr. f. Biol.*, 1895, *32*, 313–369.

Vol. XXXI, No. 6

SOME MECHANICAL FACTORS OF GASTRIC PHYSIOLOGY*

STUDY II. THE PYLORIC MECHANISM THE EFFECT OF VARIOUS FOODS ON THE EMPTYING OF THE STOMACH

By CESARE GIANTURCO, M.D.
Fellow in Radiology, The Mayo Foundation
ROCHESTER, MINNESOTA

THE PYLORIC MECHANISM

IN ATTEMPTING to explain the emptying of the stomach, some of the early investigators assumed that peristalsis travelled as far as the incisura angularis, where a sphincter contracted and divided the stomach into two pouches; the systolic contraction of the lower pouch then pushed the food into the duodenum. This supposition seemed tenable, from the mechanical standpoint, but could not be confirmed by Cannon and other observers, who studied the stomach of intact animals by means of roentgen rays. They did not find any prepyloric sphincter, and noticed that in most cases, when the antrum contracted, its content flowed back into the body of the stomach. The opening of the pylorus did not seem to be subject to any definite rule.

Then Cannon published his hypothesis that acid in the stomach is responsible for opening of the sphincter, and that acid in the duodenum is responsible for its closing. This hypothesis can be considered insufficient, first because acid foods do not leave the stomach any faster than alkaline or neutral substances, second, because the stomach of a patient with achlorhydria often empties faster than that of a normal subject, and third, because the hypothesis does not apply to fluids, to fatty substances or to gases.

More recently, Wheelon and Thomas, and McClure, Reynolds and Schwartz, studied the movements of the pylorus and found that it contracted and relaxed rhythmically. Alvarez and Zimmermann, who studied gastric peristalsis by means of cinematographic pictures of the anesthetized rabbit, dog, and cat, with the abdomen opened, found that peristaltic waves travelled along the stomach and included the pylorus.

Unfortunately, none of the investigators was able to record simultaneously all the mechanical factors that determine the passage of gastric content into the duodenum. The balloon method gave data about the contraction and relaxation of the pylorus but gave no data about the passage of food. The same objection is valid for observations made of the outside of the stomach with the abdomen open. Ordinary roentgenoscopy, after a barium meal, discloses the waves going over the stomach; it shows the passage of food through the pylorus, but it does not give any information about the condition of the sphincter when barium is not passing through it, and it does not give information in regard to the motility and tonus of the duodenum when this is empty of barium.

Attempting to overcome these difficulties I thought that the movements of the pylorus and duodenum, as well as the passage of food, could be studied roentgenographically after opaque markers had been placed in the visceral walls, and at the same time a barium meal had been given.

In fifteen cats, I outlined with lead shot, placed under the visceral serosa, the two curvatures of the stomach, the pyloric ring, and the contours of the duodenum. A month after the operation, the animals were observed roentgenoscopically to veri-

* Abstract of thesis submitted to the faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Radiology. Work done in the Institute of Experimental Medicine, The Mayo Clinic, under the direction of F. C. Mann, W. C. Alvarez and B. R. Kirklin.

a *b*

FIG. 1. A thick, black line joins shot which were placed on opposite sides of the pylorus; thinner lines join shot placed on opposite sides of the duodenum. In *a* one can follow, from the top picture to the bottom picture the opening of the pylorus; emptying occurs because the duodenum is relaxed at the same time. In *b* the pylorus relaxes as much as in *a*, but barium does not pass because of the contracted condition of the duodenum.

fy the position of the shot; it was found that only two animals could be used satisfactorily. In the others, the shot in the duodenum fell in a plane different from that of the stomach. The machine devised by Alvarez[5] for taking cinematographic roentgenograms made it possible to take records of every phase of gastric activity. Thirty films, each running for two minutes, were exposed after feeding the animals 25 c.c. of an aqueous suspension of barium. By plotting against time intervals the changing distances between corresponding shot on the two contours of the stomach and of the duodenum, tracings were obtained which were comparable to those obtainable on a kymograph. The tracings showed clearly that the pylorus contracted when a gastric wave of contraction reached it, and it relaxed when the following wave of relaxation reached it. The rate was the same as that of the gastric peristaltic waves, or from five to eight a minute. The duodenal movements were irregular in time and intensity, and occurred at a rate of about fifteen each minute. By observing the roentgenograms (Fig. 1, *a* and *b*) and marking the tracings (Fig. 2) at the times at which the emptying occurred, it appeared that barium left the stomach only when both pylorus and duodenum happened to be relaxed at the same time.

Conclusions. 1. The pylorus responds to approaching waves of peristalsis much as does the adjacent part of the pars pylorica.

2. Food leaves the stomach when the pylorus and the duodenum are relaxed at the same time.

3. Relaxation of the pylorus alone is not followed by passage of food.

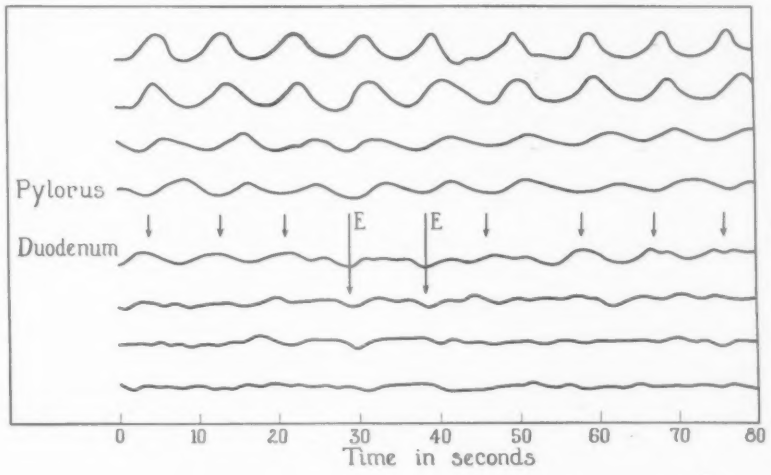


FIG. 2. The short arrows indicate pyloric relaxations not followed by exit of food because of contraction of the duodenum at the same time. The long arrows indicate pyloric relaxations followed by exit of food the duodenum is also relaxed.

THE EFFECT OF THE VARIOUS FOODS ON THE EMPTYING OF THE STOMACH

It was noticed by Cannon long ago that the emptying time of the stomach varied when different kinds of food were given. Hedblom and Cannon showed also that the consistence of foodstuffs plays an important part; that fluids leave the stomach earlier than solids, gruel more rapidly than dry potatoes, and so on. Similarly, large pieces of meat are retained in the stomach much longer than small pieces. There is no doubt that the condition in which food reaches the stomach and the facility with which it is digested play a large part in determining the emptying

time of the organ, but, in addition, other factors have to be considered.

From the studies of Cannon, Kosaka and Lim, it is well known that fats leave the stomach more slowly than proteins or carbohydrates of the same consistence. On the other hand, Carnot and Chassevant found that hypertonic solutions of salt or sugar

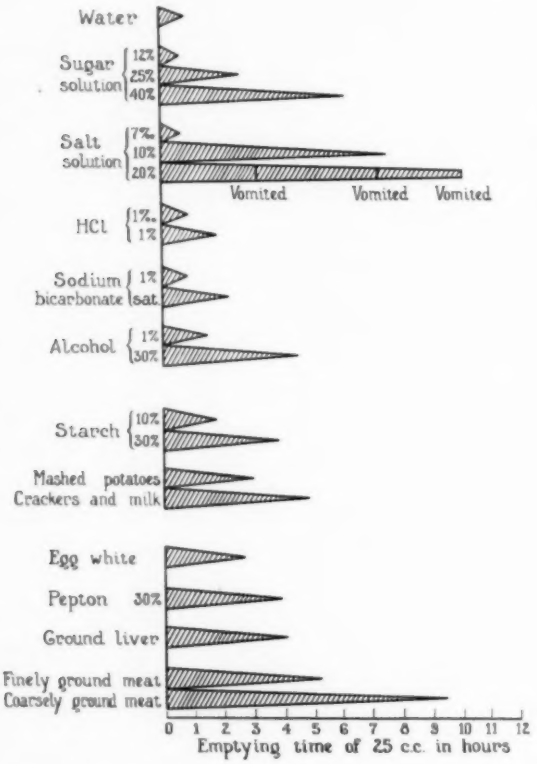


FIG. 3. The rate at which various materials left the stomach. When percentages are mentioned, the solvent was water.

remained in the stomach for a long time.

The data which may be noted in Figures 3, 4 and 5 were obtained by feeding three cats (after twenty-four hours of fasting) 25 c.c. of the different substances mixed with barium sulphate, 20 per cent by volume. The rate at which these substances left the stomach was determined by observations at short intervals with the roentgenoscope.

It was first noted that isotonic solutions of salt or sugar left the stomach in the shortest time; pure water came next. Concentrated solutions of salt and sugar remained longer in the stomach the higher their concentration. Alcohol, 30 per cent in water, left the stomach slowly. The reaction of the fluids did not seem to make much difference; both hydrochloric acid and sodium bicarbonate, when given in high concentrations, slightly slowed the emptying time of the stomach.

The statement of Cannon, that carbohydrates leave the stomach more rapidly than proteins, must be modified slightly since I found that meat in large pieces leaves the stomach more slowly than mashed potatoes, whereas finely ground liver leaves rapidly, and white of egg even more rapidly. One must take into consideration the initial consistence of both proteins and carbohydrates, as well as the facility with which they are acted on and rendered fluid by gastric juice and gastric peristalsis.

The observations of previous experimenters with regard to the delayed emptying of fats was confirmed by me. This slowing seems to be independent of the physical state of the various fats, because olive oil, which is fluid, left the stomach more slowly than lard, which is semisolid. The emptying time seemed to be proportional to the amount given, and the same was true when the oils were mixed with other foods.

The slowing was shown not only by fats but also by fatty acids and glycerin (50 per cent in water) when it was not vomited. Soaps, however, did not seem to make the emptying time longer unless they were given in very high concentration. Mineral oils lacked the slowing properties of true fats.

In conclusion, it can be said that the emptying time of different foodstuffs depends first on the physical condition, consistence, size of the particles, and so forth, and second, on the chemical nature of the substance. Fats and hypertonic solutions of salt or sugar had the most conspicuous effect in slowing the emptying of the stomach.

How Fat and Hypertonic Solutions Delay Gastric Emptying. The slow emptying of fats and hypertonic solutions has been explained as being the result of reflex closure of the pylorus. If this were true one would expect rapid exit of those substances after functional or anatomic exclusion of the sphincter. In order to test this hypothesis, sence of a pylorus. One is forced to admit, then, that this sphincter plays little part in checking the exit of fat, hypertonic solutions or water.

Several writers have claimed that there is a lack of, or a diminution in, gastric activity after ingestion of fats. In my experience, such weakening of gastric activ-

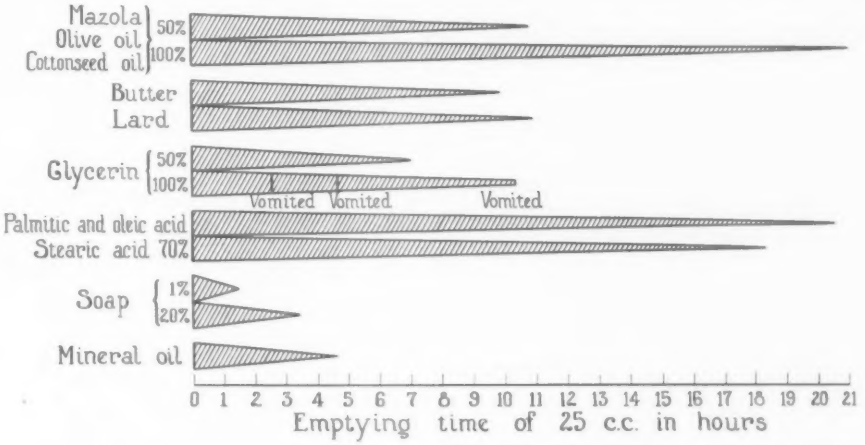


Fig. 4. The rate at which various materials left the stomach. When percentages are mentioned, the solvent was water.

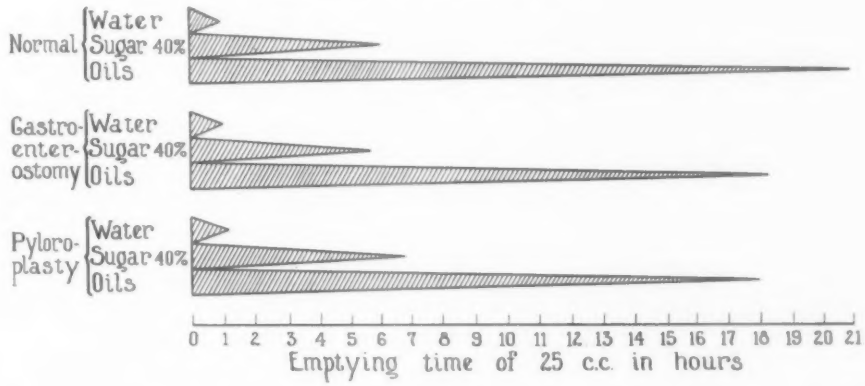


Fig. 5. The rate at which various materials left the stomach. When percentages are mentioned, the solvent was water.

four cats were studied, on two of which gastroenterostomy had been performed, and on two, pyloric excision. The emptying time of 25 c.c. of water, of oil, and of hypertonic solutions of salt and sugar in cats operated on was compared with the emptying time of the same substances in normal animals. There was little change in the emptying of all these substances in the ability is to be seen also after ingestion of hypertonic solutions of sugar or salt. What one observes is not so much a continuous lack of peristalsis as the presence of long periods in which waves are sluggish, followed by short periods of normal activity. Emptying occurred only during these periods of activity. Cinematographic roentgenograms, made at short intervals during

digestion, of cats in which the gastric and duodenal walls had been visualized by lead shot, showed that the behavior of the pylorus followed the type of peristaltic activity present; that is, the pyloric ring revealed ample contraction and relaxation during active peristalsis, whereas its movements were sluggish during the sluggish periods. The duodenum also was deeply affected; instead of showing active movements at the rate of about fifteen each minute as it does with water, its movements were tonic and short relaxations occurred only from three to five times a minute.

Conclusions. 1. Fats and hypertonic solutions induce marked changes in the activity of the gastric walls as well as of the duodenal walls.

2. The fact that the slow emptying of fats and hypertonic solutions persists after removal of the pyloric ring or after gastroenterostomy suggests that the slow emptying is due to the changes observed in gastric and duodenal motility.

REFERENCES

1. Alvarez, W. C., and Zimmermann, Arnold. Gastric wavelets. *Am. J. Physiol.*, 1926, *78*, 405–410.
2. Cannon, W. B., The acid control of the pylorus. *Am. J. Physiol.*, 1907, *20*, 283–322.
3. Cannon, W. B. The Mechanical Factors of Digestion. Longmans, Green and Company, New York, 1911, 227 pp.
4. Carnot, P., and Chassevant, A. Sur le passage pylorique des solutions de glucose. *Compt. rend. Soc. de biol.*, 1905, *57*, 1069–1072.
5. Gianturco, Cesare, and Alvarez, W. C. Roentgen ray motion pictures of the stomach. *Proc. Staff Meetings of Mayo Clinic*, 1932, *7*, 669–671.
6. Hedblom, C. A., and Cannon, W. B. Some conditions affecting the discharge of food from the stomach. *Am. J. M. Sc.*, 1909, *138*, 504–521.
7. Kosaka, T., and Lim, R. K. S. On the mechanism of the inhibition of gastric motility by fat. An inhibitory agent from the intestinal mucosa. *Chinese J. Physiol.*, 1933, *7*, 5–11.
8. McClure, C. W., Reynolds, L., and Schwartz, C. O. On the behavior of the pyloric sphincter in normal man. *Arch. Int. Med.*, 1920, *26*, 410–423.
9. Wheelon, Homer, and Thomas, J. E. Rhythmicity of the pyloric sphincter. *Am. J. Physiol.*, 1921, *54*, 460–473.

Vol. XXXI, No. 6

COLON STUDIES

VII. VARIATIONS IN FIXATION OF THE CECOCOLON; THEIR CLINICAL SIGNIFICANCE*†

By JOHN L. KANTOR, M.D., *and* SAMUEL SCHECHTER, M.D.
NEW YORK CITY

I. INTRODUCTION

PRACTICALLY all anomalies of the colon may be considered under four chief heads as follows:

1. Anomalies of length.
2. Anomalies of rotation.
3. Anomalies of descent.
4. Anomalies of fixation.

Anomalies of length and descent have formed the subjects of the first, second, and fifth studies of this series. In the present communication it is proposed to investigate, in a general way, the clinical significance of anomalies of fixation, with special reference to the cecocolon.

The process of fixation of the large intestine, whereby the ascending colon becomes attached to the areolar tissue of the posterior abdominal wall, is normally completed by about the sixth or seventh month of fetal life (Harvey). Departures from the normal development may take place in either of two directions. Thus, there may be an arrest in the process of fixation with a persistence of the primitive mesentery and increased mobility (hypermobility, hypofixation) of the ascending colon. Indeed, in extreme cases the ascending mesocolon may be continuous with the mesentery of the small intestine, resulting in the condition known as mesenterium commune.

On the other hand, there may be an abnormal progression of the fixation process, resulting in excessive adhesion (hyperfixation, hypomobility). Here the situation is more complex than in the case of hypofixation. This excess of fixation may involve the whole course of the ascending colon, or, more commonly, only certain portions of it. Harvey points out that in addition to adhesions from the gut to the parietal wall there may be bands stretching between adjacent loops of the intestine, and even localized thickenings of the ascending mesocolon. In the last named instance, obviously, hypofixation and hyperfixation exist simultaneously.

The principal points at which a normal fixation of the right colon may become accentuated are: (1) at the beginning transverse portion by the ligament of Huschke; (2) at the hepatic flexure by the right renocolic ligament of Haller; and (3) at the cecum by the parietocolic and mesenteriocolic folds. All of these structures may be regarded as causes of hyperfixation when their size and strength reaches such dimensions as to limit abnormally the natural movements of the colon. Still more distinctly atypical is the condition known as pericolic hyperfixation or Jackson's veil, in which there is obvious fusion of peritoneal surfaces normally separate. The structures causing this fixation vary in type from film-like prolongations of the peritoneum, scarcely to be observed until the intestine is distended, to distinctly thickened and strong bands of peritoneum causing acute angulation of the gut (Harvey).

Interest in the clinical aspects of variations in fixation seems to have been initiated by surgeons. It was apparently the failure to relieve right lower quadrant pain by appendectomy that first attracted at-

* Read before the American Congress of Radiology, Chicago, Ill., Sept. 25–30, 1933.

† This is the seventh of a series of articles: I. The redundant colon, Am. J. Roentgenol. & Rad. Therapy, 1924, *12*, 414–430; II. The low cecum. *Ibid.*, 1925, *14*, 207–215; III. The clinical significance of ileal stasis; its association with colitis. *Ibid.*, 1926, *16*, 1–9; IV. The roentgen diagnosis of colitis (the irritable colon). *Ibid.*, 1927, *17*, 405–416; V. The high cecum. *Ibid.*, 1928, *19*, 101–111; VI. Cecal stasis: its clinical significance and relation to proximal colon stasis. *Ibid.*, 1930, *24*, 1–20.

tention to this subject. In 1908 Wilms incriminated excessive mobility of the cecum as the cause of this symptom, whereas in the following year Jackson ascribed the same complaint to hyperfixation of the cecocolon. This naturally led to the development of two opposing schools of thought. According to one view, advocated by such men as Wilms and Klose in Germany; Waugh, McConnell and Hardman, and Carslaw in Great Britain; Coffey, Quain, Small, Beall and Aynesworth in this country, excessive mobility of the right colon is responsible for a wide variety of symptoms, ranging from right lower quadrant pain to clinical syndromes resembling ulcer, gallbladder disease, and even cancer. Various operative procedures were recommended from simple cecopexy, on the one hand, to elaborate retroperitoneal fixation of the entire cecocolon, on the other. For a critical, though on the whole favorable presentation of this type of operation the reader is referred to the paper by Houston, of Augusta, Georgia.

According to the opposite viewpoint it is fixation by adhesions that is responsible for the symptoms under discussion, and division of these adhesions, rather than additional fixation is to be recommended as the operation of choice. These ideas were advocated by Jackson in this country, and in England by Walton, and by Carson, who found that "operations to fix what should be mobile organs are not successful," and by Flint, who doubts that a mobile colon can produce more than an occasional appendicular colic. Moreover, it will be recalled that Sailer in his classic review, decided that the case for cecum mobile as a clinical entity was "not proven."

The ultra-conservative standpoint is perhaps best presented by Bedingfield in his painstaking review of the entire field of visceroptosis, when he concludes that "the physiological integrity of the colon is quite independent of its position and mobility."

A review of the above literature reveals a lack of important basic data concerning fixation of the colon. To be sure there have been available the classic anatomical contributions of Treves, Smith, and Harvey, but studies of the clinical significance of variations of colonic fixation in the living subject have been conspicuously few.* This is all the more to be deplored since modern roentgen methods have made such studies possible. In this connection may be recalled the early (1921) paper by Payne and Trahar in which several cases of normal and abnormal hepatic flexure and cecal mobility were illustrated. Barclay, in a recent study of three normal individuals, concludes that "in normal subjects the abdominal organs are not fixed and that they have a wider range of movement, both in relation to the abdominal wall and to each other, than might be expected," and expresses the hope that his "small study will do something to prick the last bubble of fixation and similar operations."

A somewhat more detailed investigation is that of McConnell and Hardman who observed cecocolic position, length and mobility in 6 normal male and 12 normal female subjects. Their findings will be discussed later in connection with our own.

II. OBJECTIVES, MATERIAL, METHODS

The present study was undertaken: (1) to determine the range of mobility of the cecocolon; (2) to establish criteria for excessive mobility and excessive fixation, and to report their incidence, and (3) to determine the clinical significance, if any, of these departures from the normal.

The material used was an unselected series of 383 cases (190 males, 193 females) in which the mobility of the cecocolon was studied. These cases were all private patients complaining of various gastrointestinal symptoms and were of the same type—being indeed a continuation of the series—as those in whom our previous colon studies were made. In addition, the results of a survey of 3,000 unselected cases of the same group were used as a basis for the general

* A brilliant exception is Wandel's work on volvulus of the cecum and ascending colon (1903).

incidence of the symptoms selected for investigation.

The roentgen method was employed to determine the degree of mobility of the cecocolon. The technique was as follows:

All tests were made at the "nine hour observation" (i.e., nine hours after the administration of a barium meal, and four hours after an ordinary meal, in a subject whose gastric emptying time was known to be five hours). The nine hour interval was chosen because the cecocolon is most clearly outlined at that time. Films were made at a distance of 27 inches in both erect and prone positions. The location of the hepatic flexure was noted in relation to the crest of the right ilium in each position. Similarly, the location of the tip of the cecum was noted in relation to the top of the right acetabulum. In plotting the position of the hepatic flexure, the topmost point, whether barium or gas filled, was taken as a landmark.

It should be pointed out that cases of non-rotation of the colon, which are, properly speaking, instances of abnormal fixation as well, cannot be studied by the methods described above. We have encountered a few such cases in our experience and although they will be mentioned in this paper to complete the presentation of the subject, their detailed description will be deferred to another occasion.

III. BASIC FINDINGS AND CRITERIA

Position of Hepatic Flexure and Cecum. As shown in Table 1, the average location of the hepatic flexure observed in the *prone* position nine hours after a standard opaque meal, is slightly over 3 inches above the crest of the right ilium. It lies about ½ inch higher in males than in females. In the *erect* posture the average position of the hepatic flexure is slightly less than 1½ inches above the ilium, being about ⅔ inch higher in males than in females (Fig. 1).

These figures compare fairly well with those of McConnell and Hardman and with those of Barclay. The former made their observations twenty-four hours after the barium meal. The interval in Barclay's cases is not stated. The Irish observers found the hepatic flexure in the prone posture to be 4 inches above the crest in males and 3 inches above in females, whereas Barclay found it "three or four" inches above this level. In the erect posture, according to the former observers, the hepatic flexure was 1½ inches above the crest in males and ¼ inch above in females. Barclay found it "an inch or more" above the crest, the sex not being stated.

The difference between our figures and those of our British colleagues may be explained either by the small number of

TABLE I

LOCATION OF HEPATIC FLEXURE AND CECUM

	Hepatic flexure		
	Position in inches above iliac crest		Excursion
	Prone	Erect	
Males	3.37	1.79	1.58
Females	2.80	1.11	1.69
Average	3.11	1.44	1.67
	Cecum		
	Position in inches above top of acetabulum		Excursion
	Prone	Erect	
Males	1.99	0.65	1.33
Females	0.99	−0.32*	1.31
Average	1.48	0.16	1.32

* Below the top of acetabulum.

TABLE II

RELATIVE INCIDENCE OF NORMAL AND ABNORMAL MOBILITY IN 383 CASES

	Hepatic flexure		
		Cases	Per Cent
Hypermobile	3 inches or over	40	10.4
Normal	between 1 and 3 inches	236	61.7
Fixed	1 inch or less	107	27.9
	Cecum	Cases	Per Cent
Hypermobile	2½ inches or over	40	10.4
Normal	between 1 and 2½ inches	177	46.2
Fixed	1 inch or less	166	43.3

cases reported by the latter, or by their choice of the twenty-four hour period for their observations, a time at which the mobility of the cecocolon may possibly be greater than at nine hours.

The average position of the cecum in the *prone* posture in our series is about 1½ inches above the top of the right acetabulum, being 1 inch higher in males than in females. In the *erect* posture the average location of the cecum is about ⅙ inch above the acetabulum, being again 1 inch higher in males than in females. It is interesting to note that in this posture (erect) the cecal tip in females averaged ⅓ inch below the top of the acetabulum (Fig. 1).

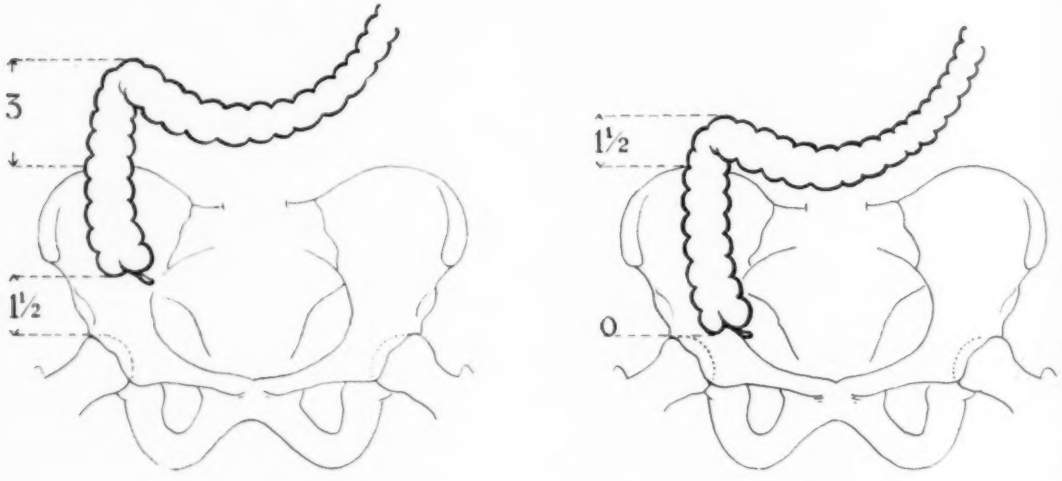


FIG. 1. Normal mobility of cecocolon nine hours after opaque meal. A. Prone. B. Erect.

Length of the Cecocolon. The average length of the cecocolon is just short of 7 inches (6.82 inches). In females it is ¼ inch greater in the prone and 1/10 inch greater in the erect posture than it is in males. The length of the cecocolon, as far as we could determine by measurement, seems to be fairly constant in both prone and erect positions. The same conclusion was reached by McConnell and Hardman. This constancy is rather surprising in view of the greater average drop in position of the hepatic flexure than of the cecum in the change from the prone to the erect posture (see below), and we are at a loss to explain it except on the assumption that our technique is not sufficiently refined to register such a difference.

Range of Mobility. The average range of mobility of the hepatic flexure in the shift from the prone to the erect posture is 1⅔ inches. This mobility is about 1/10 inch greater in females than in males. McConnell and Hardman also found a greater excursion of the hepatic flexure in females but the range of movement was considerably higher in both sexes—2½ inches for males; 2¾ inches for females—than in our series. Possible reasons for this discrepancy have already been mentioned.

The greatest vertical mobility of the hepatic flexure in our series was 4¾ inches.

The average vertical mobility of the cecum is about 1⅓ inches, or ⅓ inch less than that of the hepatic flexure. There is practically no difference (about 1/50 inch in favor of the males) between the two sexes.

The greatest vertical mobility of the cecum in our series was 4¼ inches.

As implied above, hepatic flexure mobility usually exceeds cecal mobility. This occurred in 61 per cent of our cases. It is conceivable that this difference in range of

excursion may be more apparent than real, because in observations made in the usual posteroanterior direction, the hepatic flexure probably descends in a more vertical line than does the cecum which slides from

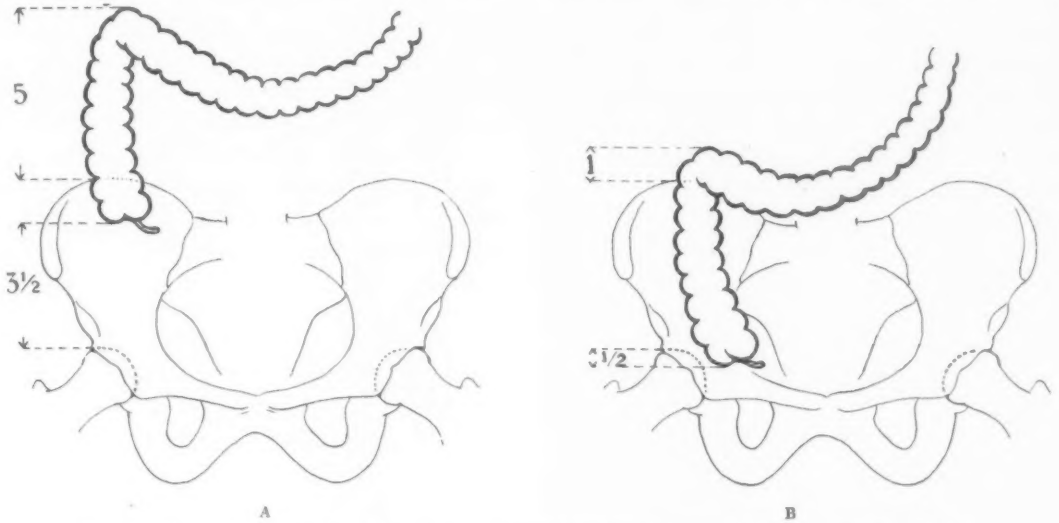


Fig. 2. Excessive mobility of cecocolon (hypofixation, hypermobility, right coloptosis) nine hours after opaque meal. A. Prone. B. Erect.

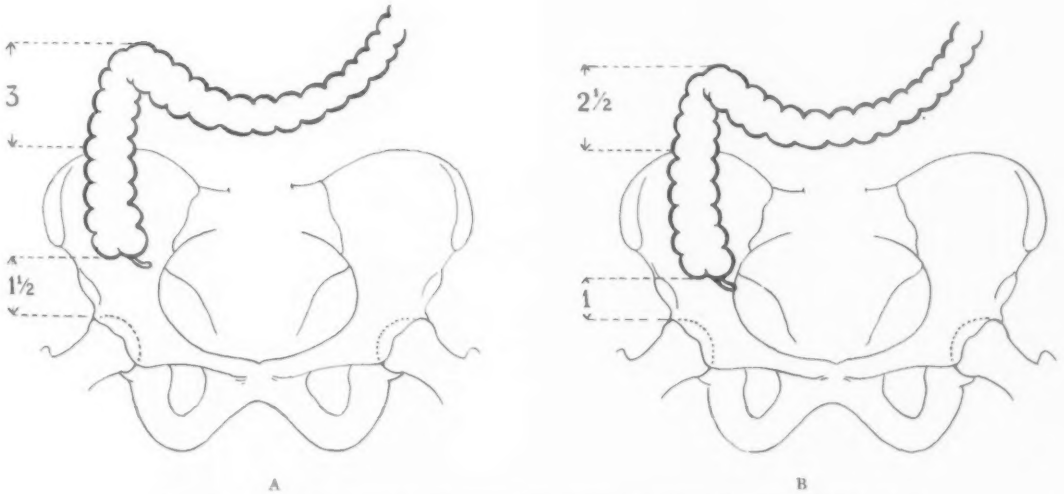


Fig. 3. Excessive fixation of cecocolon (hyperfixation, hypomobility) nine hours after opaque meal. A. Prone. B. Erect.

behind forward down an inclined plane formed by the psoas shelf.* On the other hand, there must undoubtedly be instances in which the cecum actually does move less than the hepatic flexure because of the greater relative fixation in the lower region. The opposite is also true, for in 24 per cent of cases cecal mobility exceeded hepatic flexure mobility. For the sake of completeness it may be mentioned that in 14 per cent of cases the mobility of both regions was exactly equal.

Criteria of Abnormal Fixation. For the purpose of this study excessive mobility of the hepatic flexure (hypofixation, hypermo-

* According to Coffey this inclined plane forms an angle of 51° with the horizontal and takes up 30 per cent of the weight of an organ lying upon it.

bility, right coloptosis) was arbitrarily diagnosed when the vertical excursion reached 3 inches or more. Abnormal mobility of the cecum was diagnosed when the excursion reached 2½ inches or more (Fig. 2).

Excessive fixation (hyperfixation, hypomobility) of either the hepatic flexure or the cecum was arbitrarily assumed to exist when the vertical range in mobility resulting from change in posture was one inch or less (Fig. 3).

The relative incidence of normal and abnormal mobility is shown in Table II.

IV. THE INCIDENCE OF EXCESSIVE MOBILITY (TABLE III)

Hepatic Flexure. According to the criteria utilized in this paper, excessive mobility of the hepatic flexure occurred in 40 of our 383 cases, an incidence of 10.4 per cent. It is difficult to compare these figures with the literature because similar roentgen studies of the mobility of the hepatic flexure as such have apparently not been made.

The best we can do is to review the anatomical studies in which the frequency of a persistent ascending mesocolon has been noted. Thus Treves discovered this structure in 26 per cent of 100 bodies examined for this purpose. Smith, in a series of 982 new-born infants, found an ascending mesocolon in 30 per cent and a mesenterium commune in an additional 1.7 per cent. The nearest approach to our findings are the figures of Harvey who reports a mesocolon on the right side in 13 per cent of 105 infant cadavers.

Sex. Excessive hepatic flexure mobility predominated in females over males in the proportion of 60 per cent to 40 per cent. In Harvey's smaller series 80 per cent were females, indicating, as he believes, "a later fixation of the intestine in the female."

Habitus. Hepatic flexure mobility occurred over four times as often in sthenics (47 per cent) as in asthenics (10 per cent).

Associated anomalies. The incidence of high ceca was over twice as great as normal in the hepatic flexure mobility cases (15 per cent vs. normal 6.4 per cent), and, conversely, the incidence of hepatic flexure mobility was slightly higher than normal in the high cecum series. The incidence of low ceca in the hepatic flexure mobility series was reduced slightly below the normal figures. This relative infrequency of

Table III

INCIDENCE OF EXCESSIVE MOBILITY

	Hepatic Flexure		Cecum		Cecocolon		General Incidence
	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Per Cent
General	40	10.4	40	10.4	15	3.9	
Sex							
Females	24	60.0	19	47.5	8	53.3	49.3
Males	16	40.0	21	52.5	7	46.7	50.7
Habitus							
Asthenic	4	10.0	3	7.5	1	6.6	21.0
Sthenic	19	47.0	22	55.0	9	60.0	28.6
Associated anomalies							
High cecum	6	15.0	12	30.0	4	26.6	6.4
Low cecum	5	12.5	1	2.5	0	0.	18.8
Duodenal bands	3 (in 39 cases)	7.7	3	7.5	2	13.3	4.8

hyperfixation and hyperdescent was also mentioned by Harvey. The incidence of duodenal bands was apparently slightly increased (7.7 per cent vs. normal 4.8 per cent), but this finding must not be accepted as final owing to the small number of cases (3). Besides, it is not in accord with our previous observations that sthenic individuals have a definitely low incidence of duodenal bands.

Cecum. The incidence of excessive mobility of the cecum was exactly the same as that of the hepatic flexure, namely 40 cases in 383, or 10.4 per cent. These figures correspond closely with those of Engel, Treves, and Wandel. The last named made a particularly careful survey of 640 autopsies and found that a mobile cecum, of a degree sufficient to permit kinking, torsion, or displacement, occurred in 66 cases, an incidence of 10.3 per cent.

Sex. In our series the males predominated slightly over the females in the proportion of 52.5 per cent to 47.5 per cent. These findings also are confirmed by Wandel who found an incidence of 53 per cent males and 47 per cent females.

Habitus. Sthenics show a striking predominance over asthenics in our mobile cecum cases. There were 55 per cent of the former and 7.5 per cent of the latter.

Associated anomalies. The incidence of high cecum was increased (30 per cent vs. normal 6.4 per cent) and that of low cecum diminished (2.5 per cent vs. normal 18.8 per cent). Conversely the incidence of mobile ceca was increased $3\frac{1}{2}$ times in 24 roentgen-rayed cases of high cecum and was diminished to $\frac{1}{8}$ the basic level in 82 cases of low cecum. Duodenal bands occurred in 3 cases of our mobile cecum group, but, as stated in connection with increased hepatic flexure mobility, this number is probably too small to have statistical importance.

Cecocolon. Excessive mobility of both hepatic flexure and cecum in the same subject (cecocolic hypofixation) occurred in only 15 of our 383 cases, an incidence of 3.9 per cent. This small group, as will be seen again later, occupies a position midway between that of simple hepatic flexure mobility and that of simple cecal mobility. Females predominated over males (53.3 per cent vs. 46.7 per cent), and sthenics over asthenics (60.0 per cent vs. 6.6 per cent). High ceca occurred in 27 per cent. There were no low ceca. There were

TABLE IV

INCIDENCE OF EXCESSIVE FIXATION

	Hepatic Flexure		Cecum		Cecocolon		General Incidence
	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Per Cent
General	107	27.9	166	43.3	76	19.8	
Sex							
Females	42	39.3	79	47.6	31	40.8	49.3
Males	65	60.7	87	52.4	45	59.2	50.7
Habitus							
Asthenic	20	18.7	52	31.3	18	23.6	21.0
Sthenic	34	31.7	46	27.7	22	28.9	28.6
Associated anomalies							
High cecum	3	2.8	7	4.2	4	5.2	6.4
Low cecum	28	26.1	64	38.6	28	36.8	18.8
Duodenal bands	10	10.2	17	10.7	8	10.9	4.8
	(in 97 cases)				(in 73 cases)		

2 cases of duodenal bands. In addition we have encountered 3 cases of non-rotation of the cecum. Since these cases must necessarily present the condition of mesenterium commune they should be mentioned in this connection.

V. THE INCIDENCE OF EXCESSIVE FIXATION (TABLE IV)

Hepatic Flexure. According to the criteria utilized in this paper excessive fixation of the hepatic flexure occurred in 107 of our 383 cases, an incidence of 27.9 per cent. According to Harvey, fixation in the region of the hepatic flexure may be caused either by the hepatocolic ligament (cystocolic fold, ligament of Huschke) or by the presence of the colic omentum of Haller (i.e., descent of the omentum upon the casending colon for a distance of over 2 cm.) In Harvey's 105 infants the incidence of the former structure was 18 per cent and of the latter 9.5 per cent, making a total incidence of hepatic flexure fixation of 27.5 per cent, a figure practically the same as our own.

Sex. Excessive hepatic flexure fixation predominated in males in the proportion of 60.7 per cent to 39.3 per cent. The only comparable figures in the literature are again those of Harvey. His observations refer only to the presence of the cystocolic fold which occurred in females in 60 per cent of his 20 cases.

Habitus. The incidence of sthenics and of asthenics in the group of excessive hepatic flexure fixation cases showed practically no departure from the general incidence in unselected cases.

Associated anomalies. Low ceca occurred one and one-half times as often in the hepatic flexure fixation cases as in the unselected series (26.1 per cent vs. normal 18.8 per cent), whereas high ceca occurred less than half as often. Conversely, fixed hepatic flexures occurred one and one-fourth times as often as usual in 83 cases of low cecum, and less than one-half as often in 24 cases of high cecum. The incidence of duodenal bands was twice as high as normal, and conversely fixed hepatic flexures occurred slightly more than one and one-fourth times the basic incidence in 24 cases of duodenal bands. The above findings in regard to the association of hepatic flexure fixation and cecal hyperdescent (low cecum) are confirmed by Harvey.

Cecum. Excessive fixation of the cecum occurred in 166 of our 383 cases, an incidence of 43.3 per cent. Since this figure differs somewhat from those (32 per cent) previously published by us in our studies, a word of explanation may be in order. In the previous studies observations were made only on low ceca and the criterion of fixation was restriction of cecal mobility to one inch or less in the prone posture at all observations throughout the progress of the opaque meal and before and after the evacuation of the opaque enema. In the present study all ceca were included, whether high, low or normal in descent; but the only observation made was extent of excursion on change from prone to erect position at the nine hour examination.

It is extremely difficult to compare our incidence figures with those of the literature. In the first place we cannot deny the possibility already mentioned in our section on "Range of Mobility," that owing to foreshortening unavoidable in our technique the actual mobility of the cecum may be greater than our measurements indicate, with the result that our figures for excessive fixation may be higher than they should be. In the second place, even among anatomists who work free from the limitations imposed on roentgenologists, there is little consensus of opinion as to what structures must be present to constitute pericecal hyperfixation Thus Harvey gives an incidence of 12.7 per cent for pericecal hyperfixation, and he even includes membranes that lie only on the cecum without attachment to the parieties. All these considerations do not invalidate the figures obtained by the roentgen method, since they are in every way comparable with any other roentgen data obtained by similar technique.

Sex. Excessive cecal fixation seems to predominate slightly in males, our figures being 52.4 per cent for males as against 47.6 per cent for females.

Habitus. Asthenics predominated over sthenics (31.3 per cent vs. 27.7 per cent), thus reversing the basic figures (21 per cent asthenics vs. 28.6 per cent sthenics).

Associated anomalies. Low ceca occurred more than twice as often as normal (38.6 per cent vs. 18.8 per cent) in the fixed cecum cases. The incidence of high ceca was reduced slightly below the normal figures (4.2 per cent vs. 6.4 per cent). Conversely, the incidence of fixed ceca was preceptibly increased above normal in the series of 83 low ceca, whereas it was considerably reduced below the average in 24 cases of high cecum. Duodenal bands occurred twice as often as normal in fixed ceca (10.7 per cent vs. 4.8 per cent), and conversely, fixed ceca occurred more often than normal in the 24 cases of duodenal bands (33 per cent vs. 28 per cent).

Cecocolon. Excessive fixation of both hepatic flexure and cecum in the same subject (cecocolic hyperfixation) occurred in 76 of our 383 cases, an incidence of 19.8 per cent. The incidence figures corresponded to those just cited for the hepatic flexure alone and for the cecum alone in regard to sex and anomalies. The habitus incidence was practically normal.

VI. CLINICAL SIGNIFICANCE OF EXCESSIVE MOBILITY (TABLE V)

Hepatic Flexure. General symptoms. In our 40 cases of hypermobile hepatic flexure the incidence of headaches was increased to one and one-half times the general incidence (35 per cent vs. 23 per cent). The converse relationship, however, is not true: there was no appreciable increase of hepatic flexure mobility above the basic incidence in a series of 110 roentgen-rayed headache cases. In other words, the evidence for an intimate relation between headaches and hepatic flexure mobility is by no means so clean cut as, for example, in the case of the low cecum.

The tendency to vomit seems diminished in cases of hepatic flexure hypermobility (15 per cent vs. normal 20 per cent). Conversely, hypermobile hepatic flexures occurred in less than the basic incidence (7.7 per cent vs. 10.4 per cent) in 91 roentgen-rayed vomiting cases.

Accordingly we have little reason to believe that there is any important clinical connection between either headaches or vomiting and hepatic flexure hypermobility.

Local symptoms and signs. The incidence of right lower quadrant pain was increased two and one-half times (25 per cent vs. 9.5 per cent) and that of right lower quadrant tenderness one and one-half times (27.5 per cent vs. 19.5 per cent) over their basic incidence, in our group of hepatic hypermobility cases. Conversely, hepatic flexure hypermobility was increased one and one-half times over its basic incidence in 57 roentgen-rayed cases of right lower quadrant pain and also one and one-half times over its basic incidence in 90 roentgen-rayed cases of right lower quadrant tenderness. One may therefore assume a close relation between increased hepatic flexure mobility, on the one hand, and pain and tenderness in the right lower quardant, on the other. In view of these findings it was thought of interest to investigate the incidence of appendectomy. This operation was performed one and one-half times more frequently in the hypermobile hepatic flexure cases than in the general series (25 per cent vs. 17.2 per cent). Yet the incidence of hepatic flexure hypermobility was only slightly increased above the basic level (11.1 per cent vs. 10.4 per cent) in 99 roentgen-rayed appendectomized patients. One would therefore hesitate to conclude (as it was concluded in the case of low cecum, for example) that the presence of mobile hepatic flexure is in itself responsible for an increased frequency of appendix operations.

Associated conditions. Both constipation (57.5 per cent vs. 46.5 per cent) and cecal stasis (33.3 per cent vs. 27.5 per cent) were

found to be slightly increased over their general incidence. Conversely, also, the incidence of hepatic flexure mobility was slightly increased in 182 roentgen-rayed constipation cases. In cecal stasis, however, its incidence was practically unchanged from the basic figures. "Colitis" (i.e., colonic irritability), on the other hand. showed no appreciable variation in hepatic flexure hypermobility from the basic figures; nor did the incidence of hepatic flexure hypermobility vary much from the average in 239 roentgen-rayed cases of colitis. In other words, hepatic flexure hypermobility seems, if anything, to be associated with a tendency to colonic stasis rather than to irritability.

An interesting finding was noted in regard to gallbladder disease. This occurred twice as often in mobile hepatic flexures as in the general series and four times as often as in the fixed hepatic flexures. Conversely also, the incidence of hypermobile hepatic flexures was increased about one and one-half times over the basic incidence (15.8 per cent vs. 10.4 per cent) in 19 roentgen-rayed cases of proved gallbladder disease. Unfortunately, we have here but a small number of cases—5 in the mobile, and 3 in the fixed group—but we believe that our observations are sufficiently clear-cut to be confirmed by subsequent experience. This subject will be discussed further in connection with hepatic flexure fixation.

Cecum. General symptoms. In the 40 cases of cecal hypermobility the incidence of headaches was increased above that of the basic level (30 per cent vs. 23.3 per cent). As in the case of hepatic flexure hypermobility, however, the converse was not true; there was no appreciable increase of cecal mobility in the series of 110 roentgen-rayed headache cases.

The tendency to vomit was slightly diminished in cecal hypermobility and the converse relationship was also true. There is therefore little evidence pointing to an intimate association between cecal hypermobility and either headaches or vomiting.

Local symptoms and signs. There was no increase in the incidence of either right lower quadrant pain or right lower quadrant tenderness in the series of hypermobile ceca. Conversely the incidence of hypermobile ceca was even lower than basic in 50 roentgen-rayed cases of right lower quadrant pain and in 90 cases of right lower quadrant tenderness examined roentgenologically.

The frequency of appendectomy in cecal hypermobility was the same as in the general series. Conversely, the incidence of cecal hypermobility in 99 roentgen-rayed appendectomized patients was somewhat

TABLE V

CLINICAL FINDINGS IN EXCESSIVE MOBILITY

	Hepatic Flexure (40 cases)		Cecum (40 cases)		Cecolon (15 cases)		General Incidence
	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Per Cent
Headaches	14	35.0	12	30.0	5	33.0	23.3
Vomiting	6	15.0	6	15.0	2	13.3	19.9
Right lower quadrant pain	10	25.0	4	10.0	3	20.0	9.5
Right lower quadrant tenderness	11	27.5	7	17.5	2	13.3	19.5
Appendectomy	10	25.0	7	17.5	2	13.3	17.2
Constipation	23	57.5	13	32.5	7	46.6	46.5
Cecal statis	13 (in 39 cases)	33.3	11	28.9	5 (in 14 cases)	35.7	27.5
"Colitis"	23	57.5	29	72.5	11	73.3	57.4
Gallbladder disease	5	12.5	5	12.5	3	20.0	6.1

reduced below the basic level (7.1 per cent vs. 10.4 per cent).

There is therefore no relation between cecal hypermobility and right lower quadrant pain or tenderness, on the one hand, or previous appendectomy, on the other.

Associated conditions. Constipation occurred less often in hypermobile ceca (32.5 per cent) than in the general series (46.5 per cent). Conversely, the incidence of cecal hypermobility in 182 roentgen-rayed cases of constipation was also slightly diminished (7.1 per cent vs. basic 10.4 per cent). The incidence of cecal stasis in hypermobile ceca was not appreciably altered from the basic figures, and vice versa.

On the other hand, the incidence of "colitis" was appreciably increased above the basic (72.5 per cent vs. 57.4 per cent) in cecal hypermobility. The converse finding was not so marked: the incidence of cecal hypermobility was only slightly increased above the basic (11.7 per cent vs. 10.4 per cent) in 239 roentgen-rayed cases of colitis.

From the above it may be concluded that cecal hypermobility is associated with an increase rather than with a diminution in colonic irritability, a finding opposite to that in hypermobility of the hepatic flexure.

Although gallbladder disease occurred more often in mobile ceca, mobile ceca occurred less often than the average in gallbladder disease. Hence there seems no reason to suspect any significant relationship between the two conditions.

Cecocolon. Owing to the small number of cases (15) it is impossible to draw conclusions by the use of the statistical method. Our general impression in regard to cecocolic hypermobility is that the findings concerning general and local symptoms and associated conditions are about "midway" between those for hypermobility of the hepatic flexure alone and hypermobility of the cecum alone.

It is important to note that it is only in cases of non-fixation of the cecocolon, with or without a mesenterium commune, that there can occur such acute surgical complications as volvulus of the cecocolon, ileocecal intussusception or prolapse of the cecum through a hernia. In addition to our 15 cases of hypermobile cecocolon we had 3 cases of non-rotation of the cecum. These latter, with their accompanying mesenteria commune presumably represent the most mobile arrangement of both the small and large intestine encountered in extrauterine life. None of our mobile cases, however, developed any of the acute surgical complications above mentioned.

TABLE VI

CLINICAL FINDINGS IN EXCESSIVE FIXATION

	Hepatic Flexure (107 cases)		Cecum (166 cases)		Cecocolon (76 cases)		General Incidence
	Cases	Per Cent	Cases	Per Cent	Cases	Per Cent	Per Cent
Headaches	32	29.9	62	37.4	25	32.9	23.3
Vomiting	27	25.2	52	31.3	24	31.5	19.9
Right lower quadrant pain	9	8.4	22	13.2	11	14.5	9.5
Right lower quadrant tenderness	17	15.8	37	22.3	14	18.4	19.5
Appendectomy	26	24.3	47	28.3	19	25.0	17.2
Constipation	48	44.8	77	46.4	32	42.1	46.5
Cecal stasis	31 (in 105 cases)	29.5	47 (in 165 cases)	28.4	23 (in 75 cases)	30.7	27.5
"Colitis"	71 (in 106 cases)	67.0	91 (in 164 cases)	55.5	48 (in 75 cases)	64.0	57.4
Gallbladder disease	3	2.8	9	5.4	3	3.9	6.1

VII. CLINICAL SIGNIFICANCE OF EXCESSIVE FIXATION (TABLE VI)

Hepatic Flexure. General symptoms. In cases of hyperfixed hepatic flexure the incidence of headaches was increased about one-quarter above the general incidence (29.9 per cent vs. 23.3 per cent). Conversely, there was no change from the basic figures in the incidence of fixed hepatic flexures in 110 roentgen-rayed cases of headache. Quite similar findings were observed in the case of vomiting. There is therefore no reason to assume a special association between hepatic flexure fixation and either headaches or vomiting.

Local symptoms and signs. The incidence of right lower quadrant pain was slightly, and that of right lower quadrant tenderness appreciably, diminished in hepatic flexure fixation when compared with the basic figures. More striking was the reverse relationship, i.e. the incidence of hepatic flexure fixation was considerably lower than the average in 57 roentgen-rayed cases of right lower quadrant pain and in 90 roentgen-rayed cases of right lower quadrant tenderness. Therefore, to say the least, a fixed hepatic flexure, unlike a mobile one, does not seem to predispose to right iliac pain and tenderness.

The incidence of appendectomy in hepatic flexure fixation is moderately higher than the average (24.3 per cent vs. 17.2 per cent), but the incidence of hepatic flexure fixation is less than the average in 99 roentgen-rayed appendectomized patients.

Associated conditions. There is no alteration from the basic figures in the incidence of constipation in hepatic flexure fixation and vice versa. The same holds true for cecal stasis.

On the other hand, the incidence of "colitis" in hepatic flexure fixation was 67 per cent as compared with a basic incidence of 57.4 per cent. Conversely, the incidence of hepatic flexure fixation in 239 roentgen-rayed colitis cases was also increased in somewhat similar proportion (30.9 per cent vs. 27.9 per cent).

From the above it would appear safe to conclude that hepatic flexure fixation is more intimately connected with colonic irritability than with colonic stasis.

It has already been mentioned that the incidence of gallbladder disease is considerably greater than the average in hepatic flexure mobility. In hepatic flexure fixation, on the other hand, it is less than the average (2.8 per cent vs. 6.1 per cent). The converse is also true, hepatic flexure fixation occurring in 15.8 per cent as against the basic 27.9 per cent, in 19 roentgen-rayed cases of proved gallbladder disease. Despite the small series we believe we are justified in concluding that there is no intimate relation between gallbladder disease and hepatic flexure fixation. This is all the more striking because of the rather general impression that fixation of the hepatic flexure (or at least of the proximal transverse colon in the region of the hepatic flexure) is one of the secondary signs of gallbladder disease. One must conclude, if the above data are confirmed, that congenital fixation is a far more common phenomenon than is acquired fixation in this neighborhood.

Finally, 5 cases of cholecystectomy were investigated to find out whether this operation tends to fix the hepatic flexure. Apparently it does not, for in 4 out of the 5, the hepatic flexure mobility was normal and in the fifth case it was excessive.

Cecum. General symptoms. In the fixed ceca the incidence of headaches was increased more than one and one-half times above the general incidence (37.4 per cent vs. 23.3 per cent). Conversely, the incidence of fixed ceca was increased from the basic 43.3 per cent to 55. 5 per cent in 110 roentgen-rayed headache cases. Almost similar increases were noted in the case of vomiting. In attempting to appraise the clinical significance of the above figures it is essential to bear in mind that low ceca, which are notoriously associated with a high incidence of headache and vomiting, occurred in as many as 64 of the 166 cases of fixed ceca. In order therefore to decide

what rôle in the above findings was played by the fixation rather than by the lowness of the ceca, the 64 cases of low ceca were removed from the total list of fixed ceca and the incidence of headache and vomiting was recalculated for the remainder (fixation in normal or high ceca). When this was done, the incidence of vomiting was found reduced to the basic figure, while that of headache was still found appreciably higher (33 per cent) than the general average (23 per cent).

When however, we examined a series of 78 roentgen-rayed headache cases, composed exclusively of normal or high ceca, the incidence of cecal fixation was found to be slightly lower than the basic figure. It may therefore be safely concluded that there is no significant relationship between cecal fixation *per se* and either headaches or vomiting.

Local symptoms and signs. There was a slight increase in both right lower quadrant pain and right lower quadrant tenderness in the group of fixed ceca. The converse relationship held for right lower quadrant pain but not for tenderness.

The incidence of appendectomy was increased from the basic 17.2 per cent to 28.3 per cent in the fixed cecum cases, but most, if not all, of this increase may safely be attributed to the high incidence of low ceca in this group. On the other hand, the incidence of fixed ceca was not increased above the basic level in 99 roentgen-rayed appendectomized patients. From this it may be concluded that appendectomy as such does not tend to fix the cecum.

Associated conditions. There was no alteration from the basic findings in the relation of constipation to cecal fixation, and vice versa. The same holds true for cecal stasis and for "colitis."

Just as in the case of mobile ceca, there is no relation between the incidence of gallbladder disease and cecal fixation.

Cecocolon. The findings in regard to general and local symptoms and associated conditions in cecocolic fixation were similar to those obtained in the case of fixed ceca, with the exception that the frequency of "colitis" was increased from the basic 57.4 per cent to 64 per cent. In this latter respect therefore this group resembled hepatic flexure fixation more closely than cecal fixation.

VIII. COMMENT

The above study does not appear to reveal any marked clinical disturbances associated with the usual ranges of variations in fixation of the cecocolon as a whole, or of either of its terminations (cecum, hepatic flexure) regarded separately. Such disturbances as are present seem to be adequately handled by the usual methods of conservative medical management, such as are implied in the competent treatment of the unstable colon. In none of our cases did we feel called upon to recommend surgical therapy.

We therefore believe that there is no need for operative intervention in atypical fixations of the colon except where actual evidence of intestinal obstruction is demonstrable. This would practically restrict such intervention to cases of volvulus and intussusception when excessive mobility is present, and to cases of obstruction by bands when excessive fixation is present.

The observation that hepatic flexure fixation is not associated with either gallbladder disease or gallbladder operations may be explained by the fact that in the majority of instances the fixation represents a congenital and not an acquired phenomenon. Similarly, the majority of cecal fixations may also be regarded as congenital rather than acquired in origin.

IX. SUMMARY

1. The average vertical range of mobility of the hepatic flexure is 1⅔ inches. Normal variations in mobility range from over 1 inch to under 3 inches.

2. The average vertical range of mobility of the cecum is 1⅓ inches. Normal variations range from over 1 inch to under 2½ inches.

3. Hypermobility of the hepatic flexure

may be said to exist when the vertical range is 3 inches or more, and hyperfixation when the vertical range is 1 inch or less.

4. Hypermobility of the cecum may be said to exist when the vertical range is 2½ inches or more, and hyperfixation when the vertical range is 1 inch or less.

5. Excessive mobility of either the hepatic flexure or the cecum occurs in 10 per cent of cases. Excessive mobility of the cecocolon as a whole occurs in 4 per cent of cases. Excessive mobility in general is associated with the sthenic habitus and with a high cecum.

6. Excessive fixation of the hepatic flexure occurs in 28 per cent of cases; of the cecum in 43 per cent of cases; and of the cecocolon in 20 per cent of cases. In general, excessive fixation is associated with low ceca, and with duodenal bands.

7. Clinically, hypermobility of the hepatic flexure alone is associated with right lower quadrant pain and right lower quadrant tenderness and with a tendency to colonic stasis. Hypermobility of the cecum alone is associated with increased colonic irritability. Hypermobility of the cecocolon is a prerequisite for volvulus and intussusception.

8. Clinically, hyperfixation of the hepatic flexure alone is associated with a tendency to colonic irritability. There is no evidence of association with gallbladder disease, nor does cholecystectomy tend to fix the hepatic flexure Hyperfixation of the cecum alone is apparently asymptomatic. There is no evidence that cecal fixation results from appendectomy. Hyperfixation of the cecocolon is associated with increased colonic irritability.

9. Surgical therapy does not seem indicated in the great majority of cases of variations in fixation of the cecocolon. This form of treatment should be reserved for cases in which there is evidence of intestinal obstruction.

REFERENCES

1. AYNESWORTH, K. H. Pathological consequences of the congenitally ptosed right colon; surgical treatment in selected cases. *Am. J. Surg.*, 1929, *7*, 358–368.
2. BARCLAY, A. E. The mobility of the abdominal viscera. *Quart. J. Med.*, 1932, *1*, 257–276.
3. BEALL, F. C. Deficient fixation of the right colon. *South. M. J.*, 1926, *19*, 560–565.
4. BEDINGFIELD, H. Visceroptosis and Allied Abdominal Conditions Associated with Chronic Invalidism. Oxford University Press, London, 1930.
5. CARSLAW, R. B. Right-sided visceroptosis; an estimate of the importance of abnormal mobility and prolapse of the ascending colon and caecum in the causation of various abdominal conditions, based on observations in a series of 242 cases treated by right colopexy. *Brit. J. Surg.*, 1928, *15*, 545–604.
6. CARSON, H. W. Symposium on indigestion. 1. The surgical aspect. *Post-Grad. Med. J.*, 1926, *2*, 33–35.
7. COFFEY, R. C. The principles underlying the surgical treatment of gastrointestinal stasis, due to causes other than strictural or ulcerative conditions. *Surg., Gynec. & Obst.*, 1912, *15*, 365–429.
8. COFFEY, R. C. Relation of right-sided abdominal pain to right-sided disease. *J. Am. M. Ass.*, 1923, *81*, 900–904.
9. DUVAL, P., ROUX, J. C., and BÉCLÈRE, H. The Duodenum. C. V. Mosby, St. Louis, 1927.
10. FLINT, E. R. Observations on the mobile ascending colon. *Lancet*, 1921, *1*, 903–905.
11. HARVEY, S. C. Congenital variations in the peritoneal relations of the ascending colon, cecum, appendix, and terminal ileum. *Ann. Surg.*, 1918, *67*, 641–686.
12. HOUSTON, W. R. The mobile right colon. *J. Am. M. Ass.*, 1929, *93*, 766–768.
13. KANTOR, J. L. The unstable colon. *South. M. J.*, 1932, *25*, 29–37.
14. KLOSE, H. Klinische und anatomische Fragestellungen über das Coecum mobile. *Beitr. z. klin. Chir.*, 1909, *63*, 711–741.
15. MCCONNELL, A. A., and HARDMAN, T. G. Abnormalities of fixation of the ascending colon; relation of symptoms to anatomical findings. *Brit. J. Surg.*, 1923, *10*, 532–537.
16. MORLEY, J. Clinical manifestations of the mobile proximal colon. *Brit. M. J.*, 1920, *2*, 542.
17. PAYNE, R. A., and TRAHAR, F. C. Developmental rests in cecum and ascending colon and their roentgen ray diagnosis. AM. J. ROENTGENOL., 1921, *8*, 643–653.
18. QUAIN, E. P. Pathogenic ptosis of the right colon. *Arch. Surg.*, 1923, *6*, 638–652.
19. QUAIN, E. P. Contribution to the diagnosis and treatment of right coloptosis and its complications. *Am. J. Surg.*, 1924, *38*, 193–198.

20. Quain, E. P. Colofixation in "chronic appendicitis," based on study of late results in 96 patients. *Am. J. Surg.*, 1929, *7*, 259–265.
21. Sailer, J. Cecum mobile. *Am. J. M. Sc.*, 1912, *143*, 157–172.
22. Small, A. B. Developmental errors in fixation of ascending colon, reporting 190 colopexies. *South. M. J.*, 1924, *17*, 853–865.
23. Smith, G. M. A statistical review of the variations in the anatomic positions of the caecum and the processus vermiformis in the infant. *Anat. Rec.*, 1911, *5*, 549–556.
24. Treves, F. The Anatomy of the Intestinal Canal and Peritoneum in Man. H. K. Lewis, London, 1885.
25. Walton, A. J. Clinical aspects of visceroptosis, *Brit. J. Surg.*, 1915–1916, *3*, 185–218.
26. Wandel, O. Ueber Volvulus des Coecum und Colon ascendens. *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1903, *11*, 39–79.
27. Waugh, G. E. The morbid consequences of a mobile ascending colon, with a record of 180 operations. *Brit. J. Surg.*, 1920, *7*, 343–383.
28. Wilms, M. Fixation des Coecum mobile bei Fällen von sogenannter chronischer Appendicitis. *Centralbl. f. Chir.*, 1908, *35*, 1089–1091.

MYOCARDIAL CALCIFICATION*

By JOHN J. MOORE

Major, Medical Corps, U. S. Army, Director, Department of Roentgenology, Army Medical School, Army Medical Center

WASHINGTON, D.C.

ROENTGENOLOGIC literature on the demonstration of myocardial calcification during life, and in which the findings have been subsequently confirmed by necropsy, is extremely meager. In contrast to myocardial calcification, roentgenologic literature on pericardial calcification is quite abundant.

Scholz,[5] in 1924, reported the first case on record in which calcium deposits within the heart wall itself, without involvement of the pericardium, were demonstrated roentgenographically intra vitam, and in which the roentgen diagnosis was confirmed at necropsy.

The cases published by Groedel[3] and LeWald[4] were not proved by necropsy and the cases reported by Simmonds[6] concerned roentgenograms of autopsy specimens.

A review of the literature from 1925 to 1930 reveals no cases of myocardial calcification in which roentgen diagnosis was made and subsequently proved by autopsy.

We wish to present the second case for record. The clinical brief is as follows:

A. M., aged sixty, white male, born in Palestine. Admitted to Letterman General Hospital, San Francisco, California, November 15, 1929, at 5:00 P.M., and died at 11:25 P.M.

Family History. Unessential.

Past History. Former blacksmith. Does not use alcohol; moderate user of tobacco. He had malaria in 1899 and in 1914. Has had "heart trouble" since 1918. Patient states that he had precordial pain of sharp, nonradiating type, which was brought on by exertion and prevented his doing heavy work. He has had three previous admissions to this hospital.

First admission: October 23, 1927, to Ear, Nose and Throat Ward. Diagnosis: Sinusitis, chronic, suppurative, ethmoidal and maxillary, bilateral. Treatment: Antrum puncture. Heart was normal on physical examination. Blood pressure 110/60. Discharged improved, October 31, 1927.

Second admission: June 30, 1928, to Ear, Nose and Throat Ward. Recurrence of previous sinus trouble. Treatment: Radical operation. Heart normal on physical examination. Blood pressure 135/80. Routine laboratory tests negative. Discharged cured, July 13, 1928.

Third admission: September 10, 1929, to general medical ward. With complaint of pain in left shoulder which was aggravated by motion. States his family physician's diagnosis was neuritis. Roentgenograms of left shoulder were negative for arthritic changes. The cause of the condition was not determined at this time. Incidentally he was sent to the Roentgen Department for cardiac studies because of precordial pain.

Roentgen Examination. September 21, 1929. Roentgenoscopy and films, posteroanterior, left lateral and left anterior oblique views. An ovoid area of calcification lies in the wall of the left ventricle and extends almost into the apex. It measures roughly 7×6×5 cm. Its border is ring-like and its center has a basket-weave appearance. Just above its superior border is a ring-like homogeneous area of calcification which is interpreted as being in the region of the mitral valve. No calcification is thought to be present in the pericardium.

Impression: (1) myocardial calcification, left ventricle; (2) endocardial calcification, left ventrical.

The electrocardiographic findings were interpreted as chronic myocarditis. The patient was to be examined by cardiovascular section but desired to be discharged. Diagnosis on discharge, September 25, 1929, is as follows: (1) observation arthritis, left shoulder, not found; (2) pansinusitis, chronic, bilateral, old; (3) myocarditis, chronic, severe, manifested by electrocardiographic findings; (4) calcification of myocardium demonstrated by roentgen examination; (5) arteriosclerosis, generalized, moderate.

Present Illness. Patient was admitted to

* Published with the permission of the Surgeon General, United States Army, who assumes no responsibility for any of the opinions expressed.

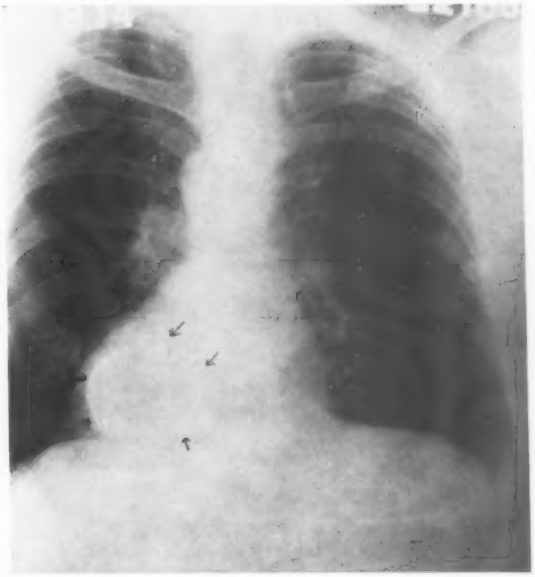


Fig. 1. Posteroanterior view. Note calcification in wall of left ventricle and a small denser calcification above in the endocardium.

cardiac ward as an emergency case. He was first seen at 6:00 P.M., November 15, 1929. He complained of severe pain in precordial region. Pulse irregular. Blood pressure 78/55. After the administration of adrenalin and morphine

Fig. 2. Left lateral view. Note location and appearance of the two areas of calcification.

Fig. 3. Oblique view, showing extension of calcification into apex of heart.

Fig. 4. Posterior aspect of heart. Note bulging of calcified portion of wall with fibrous strands of adhesion attached.

pulse became stronger. Rate 120. Temperature 97.6° F.; respiration 50.

Progress Notes. 9:00 P.M. Patient again practically pulseless. Blood pressure could not be obtained. Prognosis dubious. Died at 11:25 P.M.

The autopsy findings, which pertain to heart and aorta, are as follows:

Fig. 5. The posterior wall of left ventricle sawed open to expose the internal aspect of the calcified mass; the calcified posterior leaflet of mitral valve; chordae and papillary muscles have been divided.

Pericardium. There was no excess fluid present. The left external and posterior surface of the pericardium was adherent to lower posterior surface of heart by several short, dense strands. No calcification was present.

Aorta. Some atheromatous softening was present. Calcification was scanty.

Heart. Weighed 545 grams. The wall of the left ventricle bulged outward and contained a large deposit of calcium within it. This followed the curve of the ventricle wall and extended to about 2 cm. of the anterior boundary between the ventricles. It extended from the auriculoventricular groove downward to within 2 cm. of the apex. Roughly this mass measured 8 cm. horizontally, 7 cm. vertically and in thickness was almost 5 cm. There are also calcium deposits in left ventricular cavity involving the papillary muscle, chordae tendinae and leaflet of the mitral valve. The right coronary artery shows much sclerosis of its walls and several points of extreme narrowing of the lumen. The left coronary artery, in its anterior descending branch, also shows much sclerosis and some points of extreme narrowing. The other branch of left coronary could not be followed. The myocardium, except in its calcified areas, appeared unusually normal.

Anatomical Diagnosis. Severe sclerosis of coronary arteries with occlusion of one branch of left; myocardial calcification, extensive and advanced, left ventricle wall; fixation of mitral valve by thickened chordae tendinae; sclerosis of mitral valve; moderate sclerosis of aortic valve; few adhesions to parietal pericardium posteriorly; calcium deposits in choroid plexuses; slight pleural effusion; few adhesions at each apex; fibrosis of spleen; fibrosis of pancreas moderate passive congestion and slight cirrhosis of liver; chronic interstitial nephritis; moderately advanced; arteriosclerotic aortitis, moderately advanced.

Cause of Death. Coronary sclerosis with occlusion of one branch of left. Contributory—calcification of myocardium, wall of left ventricle.

DISCUSSION

It is generally accepted that the deposition of calcium salts within the heart takes place only in dead or markedly deteriorated, never in healthy tissues. Attention is invited to the autopsy findings in our case of obliteration of the left coronary artery, which was also present in the cases reported by Burns,[2] Askanazy,[1] and Scholz.

In passing it is interesting to note that here, as in the cases of Simmons[7] and Scholz, the patient was able to carry on a moderately bearable life, notwithstanding the fact that a large portion of the left ventricle had been rendered practically useless.

The differential roentgen diagnosis of myocardial calcification and pericardial calcification should not prove difficult in the larger deposits because in the former the calcification appears as rounded, irregularly diffused shadows which can be demonstrated to be within the heart itself

by the taking of roentgenograms at different angles, whereas in the latter the band-like shadows are seen just outside the heart shadow, and it will also be noted that the calcified pericardium forms almost a right angle with the diaphragm at its posterior attachment. Finally, the importance of the use of the grid for both roentgenoscopy and roentgenography is emphasized to bring out tissue differentiation.

REFERENCES

1. Askanazy. Ueber Kalkmetastase und progressive Knochentrophie. Festschr. z. Feier. d. 60. Geburts. v. M. Jaffe, 1901, 187.
2. Burns, A. Observations on Some of the most Frequent and Important Diseases of the Heart. Bryce and Co., Edinburgh, 1809, p. 194.
3. Groedel, F. M. Erste Mitteilung über die Differenzierung einzelner Herzhöhlen im Röntgenbilde und den Nachweis von Kalkschatten in der Herzsilhouette intra vitam. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1910–1911, *16*, 337–341.
4. LeWald, L. T. Proceedings German Medical Society of New York City, 1924.
5. Scholz, Thomas. Radiographic demonstration of calcification of the myocardium during life. *J. Radiol.*, 1924, *5*, 131–133.
6. Simmonds, M. Ueber den Nachweis von Verkalkungen am Herzen durch das Röntgenverfahren. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1908, *12*, 371–373.
7. Simmons, S. F. A case of ossification of the heart. *Med. Communications, London*, 1783, 228.

A CASE OF SPONTANEOUS PNEUMOTHORAX IN AN INFANT, WITH COMPLETE RECOVERY

By GEORGE S. REITTER, M.D.
Radiologist, Orange Memorial Hospital
ORANGE, NEW JERSEY

J. M., white, male, aged eighteen months, only child, was admitted to the hospital on Dr. von Hofe's service January 24, 1933.

Family History. Negative.

Past History. The patient had pertussis at the age of six months and possibly bronchitis at fourteen months. General health good.

Present Illness. For several days prior to his admission the patient had a cough and on Sunday, January 22, 1933, he became worse—fretful and short of breath. He vomited twice on January 23.

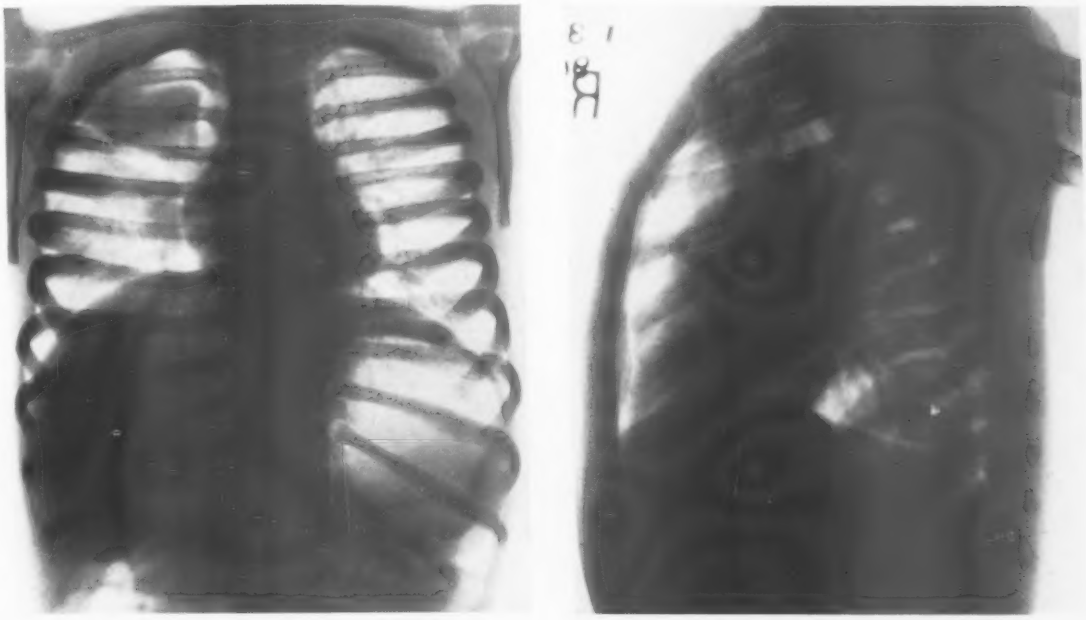
He was brought to the accident room on January 24 at 4:10 A.M. Examination revealed rapid, labored respirations with an expiratory grunt, flaring of the alae nasi, contraction of the accessory muscles on respiration, and retraction of the suprasternal notch and intercostal spaces. The child was admitted with a provisional diagnosis of a foreign body in the lungs or an enlarged thymus.

Clinical Course. Soon after admission he was sent to the Roentgen Department for examination. A diagnosis of spontaneous pneumothorax was made.

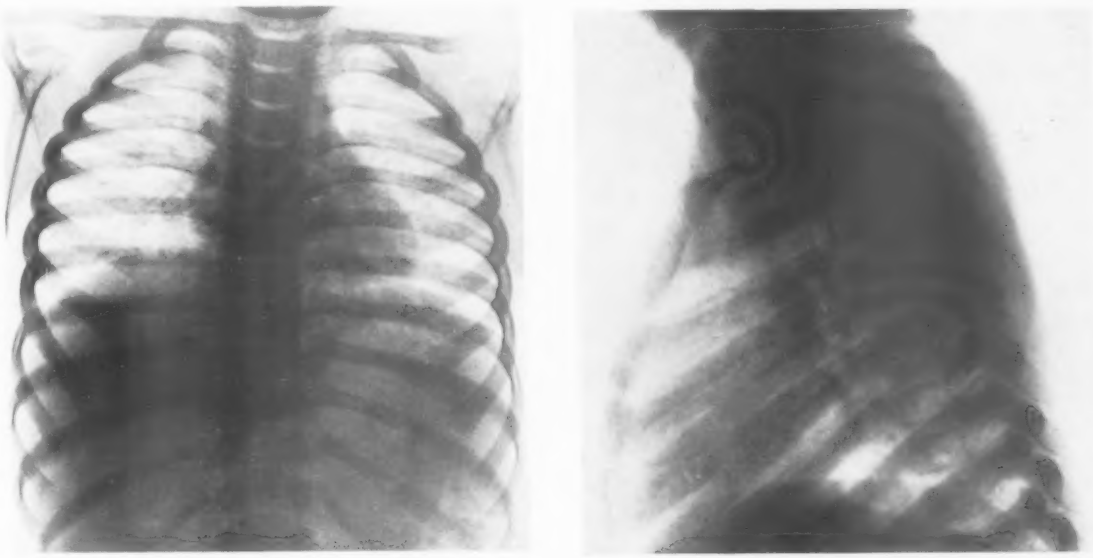
Figures 1 and 2 show emphysema of the soft tissues of the neck on both sides extending from the right apex of the lung. A pneumothorax is present on the right side extending along the outer border of the first two interspaces of the upper lobe and the entire outer border of the lower lobe. The heart is displaced slightly to the left.

FIGS. 1 and 2. Show emphysema of the soft tissues of the neck on both sides extending from the right apex of the lung. A pneumothorax is present on the right side extending along the outer border of the first two interspaces of the upper lobe and the entire outer border of the lower lobe. The heart is displaced slightly to the left.

On January 25 a temperature of 104°F. was reached with respirations 60. January 30—No air in the right upper lobe. Little change clinically. Laboratory reports of blood, urine, etc., showed nothing unusual. Mantoux test, negative.



Figs. 3 and 4. The emphysema in the left side of the neck is still present. The pneumothorax on the right side has extended from the diaphragm to the apex with a complete atelectasis of the upper lobe. The heart is no longer displaced to the left.



Figs. 5 and 6. There is a clearing of the emphysema of the soft tissues outside of the chest. A slight pneumothorax is still present on the right side along the outer lower and possibly middle lobes. The remainder of the lungs is clear.

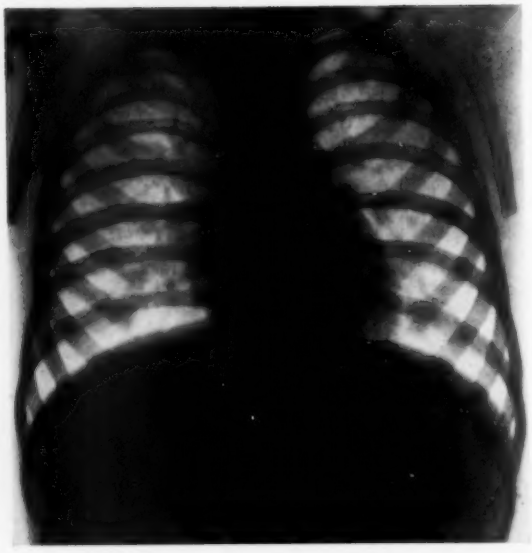


Fig. 7. The pneumothorax has entirely disappeared. (Roentgenogram taken six weeks after the original.)

The patient was transferred to the New York Medical Center on February 1 for observation. He showed general improvement and was readmitted to the Orange Memorial Hospital on February 15. Signs of a bronchitis developed with a spiking temperature reaching 105°F. on February 18. This gradually subsided. The child continued to improve and was discharged on March 11. He has been followed in the clinic and was last seen on July 25. Respirations were normal and general condition was good.

Several subsequent roentgen examinations were made which show interesting changes in the lungs.

Figures 3 and 4 were taken on January 30 and show the emphysema in the left side of the neck still present. The pneumothorax on the right side has extended from the diaphragm to the apex with a complete atelectasis of the upper lobe. The heart is no longer displaced to the left.

Figures 5 and 6 were made on February 16 and show a clearing of the emphysema of the soft tissues outside of the chest. A slight pneumothorax is still present on the right side along the outer lower and possibly middle lobes. The remainder of the lungs is clear.

Figure 7 was taken on March 1, six weeks after the original, and shows that the pneumothorax has entirely disappeared.

I wish to thank Dr. Frederick H. von Hofe in charge of the Pediatrics Department for permitting the publication of this case.

Vol. XXXI, No. 6

TERTIARY SYPHILIS OF THE ESOPHAGUS

WITH REPORT OF A CASE RECOGNIZED ROENTGENOLOGICALLY

By LESLIE F. WILCOX, M.D.
Assistant Roentgenologist, Harper Hospital
DETROIT, MICHIGAN

TERTIARY syphilitic lesions of the esophagus are rare manifestations of the disease. Guisez,[2] in 1924, stated; "La syphilis n'aime pas l'oesophage." Guyot,[3] in 1931, in an excellent monograph on syphilis of the esophagus, reviewed the cases in the literature. He collected 55 cases and added 2 personal cases, making a total of 57. The diagnosis of the two cases which he reported was confirmed at autopsy and a careful microscopic study of the lesion was made in each case. His work added much to the knowledge of the histopathological changes in the tissues of the esophageal wall in tertiary syphilitic esophagitis.

Guyot[3] stated that one may consider as authentic those cases which were verified at autopsy, of which there was a total of 11 and probably also those which, recognized by the esophagoscope, have been cured by specific treatment, of which there was a total of 15 cases. He thus would imply that many cases reported as this condition may not have been accurately diagnosed.

Many of the authors who have reported cases of syphilitic esophagitis have given a summary of the earlier cases and these need not be repeated here.

Tertiary syphilis of the esophagus occurs in both males and females. It has been stated that males are more often affected by the lesion in this organ of the body but it would seem from a review of the cases that it occurs equally as often in the female. The lesion occurs in adult life, during the age period in which syphilis is most commonly acquired.

The pathology of the lesion of the esophagus is similar to the disease elsewhere in the body. The pathological status in a certain case will depend upon the time, in its clinical course, at which that case may be observed.

The syphilitic process reaches the esophagus by one of two ways: (1) A gumma develops primarily in the wall of the esophagus, usually in the submucosa. (2) There is direct extension of the syphilitic process from a neighboring organ or tissue such as the aorta, a mediastinal gland, the lung parenchyma or the sternum. This is the more common of the two invasions.

Having thus reached the esophagus the process may then terminate in the esophageal wall in one of three ways, which are: (1) Involution by fatty degeneration and complete healing without ulceration or any appreciable fibrosis and contraction. (2) Necrosis of the tissues and extension to the mucosa with ulceration. If healing takes place without extensive involvement there will result fibrous tissue which contracts and produces a rather localized stenosis of the esophageal lumen. (3) The process may be more chronic in its progress and invade the tissues of the wall of the esophagus in a diffuse manner, encircling the lumen in the greater part of its length. There may be sclerosis and superficial ulceration. Healing of this diffuse process results in a very marked narrowing of the esophagus with inability to swallow almost all solid food.

In chronic diffuse esophagitis the inflammatory process is found to have invaded all the four coats of the wall of the esophagus. The parenchyma of the various coats is destroyed and fibrous tissue, greatly infiltrated with lymphocytes and plasmocytes, takes the place of the muscle fibers and the epithelial cells of the glands. The lymphocytes accompany chiefly the smaller blood vessels and in these vessels there is a proliferation of the intima.

The symptomatology also depends upon

the stage at which the lesion is encountered. Early, there is slight difficulty in swallowing solid food. As the stenosis of the lumen of the esophagus increases, the dysphagia increases, and it may become difficult to swallow fluids. The onset of the dysphagia is gradual and there is usually a history of its having been present at least several months. There may be a sensation of retrosternal heaviness or pressure following meals but there is rarely pain, though this may be present in the late stages during the act of swallowing. Loss of weight soon accompanies the dysphagia and in the later stages there is definite anemia and cachexia. Regurgitation of food may occur relatively late when there is almost complete stenosis.

In arriving at a diagnosis of tertiary syphilis of the esophagus the history is of great benefit. The other diseases which produce similar symptoms must be excluded. The presence of suspicious lesions in adjacent organs or in the distant parts of the body that suggest lues may give one a clue. Careful serological tests should be made, and a positive Wassermann reaction, while not conclusive proof of the lesion in the esophagus being luetic, is at least presumptive evidence. The roentgenoscopic examination with a thick opaque meal, with a succeeding roentgenographic study is of great importance. The luetic condition may be recognized and the other lesions excluded and the extent of the esophageal involvement determined as a guide to treatment.

There is no characteristic roentgen appearance of diffuse syphilitic esophagitis with stenosis but there are several features which are more or less peculiar to this condition alone. Observed roentgenoscopically, the walls of the esophagus will be quite rigid, there being very little change in the general contour or caliber of the barium column as the opaque bolus passes through the area of narrowing and the barium will have a finely irregular outline. In carcinoma, if it is extensive enough to produce rigidity of the walls for a considerable distance, there will almost always be a pronounced and coarse irregularity with also more irregular canalization. In a spasm of a segment of the esophagus, the contour will usually be smooth. An extensive stricture produced by caustics with irregular ulceration and contraction may be the most difficult of the various lesions to differentiate from the diffuse syphilitic stenosis, though here the history should be of importance.

When a gumma is present in the esophageal wall it may be impossible to make a diagnosis roentgenologically or with the esophagoscope. Esophagoscopy should always be carried out and particularly in those cases in which a diagnosis cannot be made by the other methods, and a biopsy performed where there is ulceration or tumor formation. In a suspected case of syphilitic esophagitis, the therapeutic test of improvement under thorough specific treatment will usually confirm the diagnosis.

The prognosis depends on the general condition of the patient, on the presence or absence of luetic lesions in adjacent organs, on the extent and degree of stenosis of the esophagus and on the possibility of dilatation of the esophageal canal.

Treatment is directed along two lines: (1) Antisyphilitic treatment and a building up of the patient's general condition. (2) Esophageal treatment to relieve the stenosis which accompanies the fibrosis and contraction in the healing of gumma or the diffuse ulceration in syphilitic esophagitis. This is accomplished by frequent and persistent instrumental dilatations with metal dilators. Abel[1] states that the first dilatation should be done under direct vision and subsequently if there are no contraindications it may be performed blindly. The progress of the dilatation should be followed by several roentgenoscopic examinations. It may be necessary to continue treatment over a period of one or two years.

CASE REPORT

F. B., female, white, American, aged twenty-five, married, entered the Out-Patient Department of Harper Hospital, November 5, 1931, with the complaint of difficulty in swallowing which had been progressive for the preceding six weeks. She was then having difficulty in swallowing both solid and liquid foods with occasional vomiting immediately after taking small quantities. She also complained of a swelling over the upper sternal region which had been present for ten months. She weighed 120 pounds and had lost 5 pounds during the six weeks prior to her visit to the Hospital.

She was referred to the roentgenological department for examination of the chest and the esophagus with a provisional diagnosis of one of several lesions: esophageal diverticulum, substernal thyroid, aortic aneurysm or mediastinal tumor.

We observed the rather marked prominence of the manubrium and that it was only slightly tender and did not pulsate. The roentgenoscopic examination of the chest was essentially negative. On examining the esophagus roentgenoscopically with a thick acacia-barium mixture there was noted definite narrowing of the lower esophagus from a point slightly below the level of the tracheal bifurcation to its cardiac end. The outline of this portion of the esophagus was finely irregular and the walls were rigid, there being no peristalsis in this portion. The esophagus above this level was only slightly increased in caliber. Roentgenographic study of the esophagus filled with the opaque mixture revealed irregular contour of the barium column from the clavicular level to the cardiac end. There was very definite narrowing from a point slightly below the tracheal bifurcation to its lower end and slight dilatation of the upper esophagus (Fig. 1). Roentgenographic study of the manubrium sterni revealed increased thickness of this structure with irregular density of the bone and limited periosteal proliferation, with a similar change in the sternal ends of both clavicles. The body of the sternum was not involved. We concluded that the changes in the manubrium sterni were produced by the same process which had involved the esophagus and that it was a luetic infection. A Wassermann test was requested.

The patient had been married ten years, the husband was living and well and there was one child living and well. There had been one miscarriage. She denied any venereal disease but later admitted extramarital relations over the last ten years.

The laboratory findings were as follows: urinalysis, negative; blood Wassermann four plus and blood Kahn one plus.

The diagnosis made was tertiary syphilis of the esophagus, the manubrium sterni and the sternal ends of the clavicles. The patient was referred to the dermatological section for antiluetic treatment and esophagoscopy with dilatation of the esophagus was advised.

FIG. 1. Roentgenogram showing tertiary syphilitic involvement of the esophagus from the clavicular level to the cardiac end with stenosis of the lower two-fifths. The opaque column in the involved portion is finely irregular in contour.

Follow-up Record. Intensive antisyphilitic treatment was given beginning November 17, 1931, and this has been continued up to January, 1933, using mercury succinimide, potassium iodide, neosalvarsan and bismuth.

April 2, 1932, re-examination of the esophagus roentgenologically revealed increased caliber of the lower esophagus without dilatation of the upper esophagus and the opaque meal passed more readily through to the cardiac end (Fig. 2). The patient felt very much improved, was able to swallow solid food easily and had gained several pounds in weight. The blood

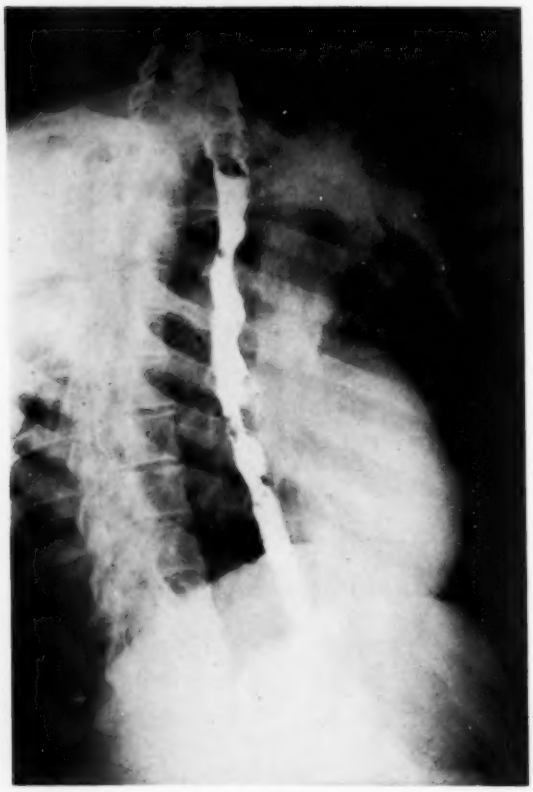


FIG. 2. Nineteen weeks after antisyphilitic treatment was started. There is increased caliber of the lower two-fifths of the esophagus.

Wassermann was still four plus. Esophageal dilatation was again advised.

April 14, 1932, she was first seen in the nose and throat division of the Out-Patient Department and because she stated she could swallow solid food, esophagoscopy was advised against. May 2, 1932, esophageal dilatation was again advised against.

August 30, 1932, blood Wassermann was still four plus.

January 5, 1933, blood Wassermann was four plus but patient stated she was almost free from discomfort in swallowing and had gained another few pounds in weight. The manubrium sterni was much less prominent and there was only slight tenderness over its lower portion. Roentgen study of the esophagus with the opaque meal revealed more nearly normal peristaltic action, only limited narrowing of the lower esophagus and no delay in the passage of the bolus (Fig. 3).

Comment. This is a case of tertiary syphilis of the esophagus in which we believe the lesion was primarily in the manubrium sterni and which reached the esophagus by direct extension through the mediastinal tissues. It was recognized roentgenologically and the diagnosis confirmed by the serological studies and the therapeutic test of improvement under thorough specific treatment. Instrumental dilatation of the contracted lumen of the esophagus was not carried out due to the belief by several consultants that it was unnecessary. The patient was seen earlier after the onset o the dysphagia than most cases and no instrumental dilatation of the esophagus has been done, yet there has been much improvement under antisyphilitic treatment. However, dilatation may yet be necessary.

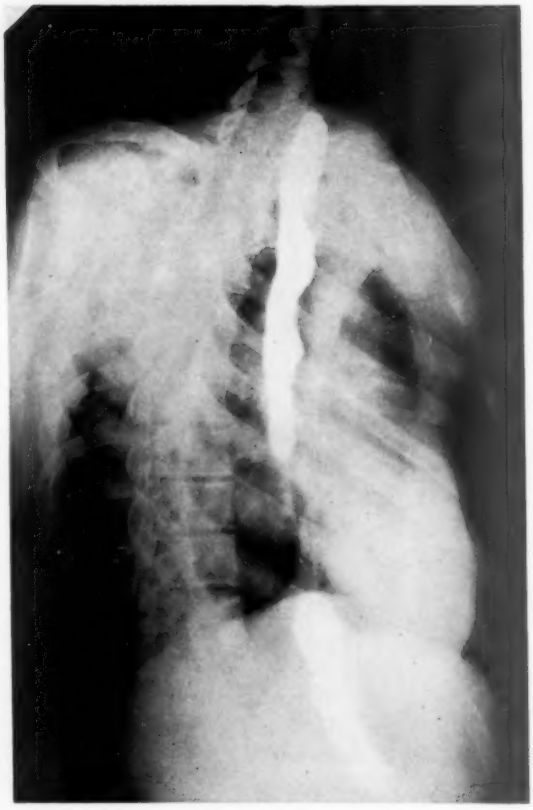


FIG. 3. Fifty-nine weeks after antisyphilitic treatment. Increased caliber in the lower esophagus and the contour is becoming quite smooth throughout. Roentgenoscopically the lower esophagus was well filled and of almost normal caliber.

SUMMARY

1. Tertiary syphilis of the esophagus is relatively rare.

2. It usually terminates by a stenosis of the lumen of the esophagus and as much as one-half or more of its length may be involved.

3. The chief symptom is dysphagia which accompanies the slowly developing stenosis.

4. Roentgenological study is of the greatest importance in the diagnosis, and serological tests and esophagoscopy should always be part of the examination.

5. Intensive antiluetic treatment must be carried out.

6. Owing to the marked fibrosis in the healing of the lesion, the stenosis is more or less permanent and instrumentation may have to be resorted to, over a long period of time, to dilate the involved portion of the esophagus.

REFERENCES

1. Abel, A. L. Syphilis of the oesophagus. *Lancet*, 1928, *2*, 441–443.
2. Guisez, J. De la syphilis de l'oesophage. *Bull. l'Acad. de méd.*, 1924, *91*, 483–486.
3. Guyot, R. La syphilis de l'oesophage en particulier au point de vue anatomo-pathologique. *Ann. d'oto-laryng.*, May, 1931, 505–526.
4. McMahon, F. B. Luetic obstruction of the oesophagus. *Surg., Gynec. & Obst.*, 1923, *37*, 141–143.
5. Moore, A. B. Benign lesions in the cardiac portions of the esophagus and stomach. *J. Am. M. Ass.*, 1930, *94*, 12–15.
6. Myerson, M. C. Syphilis of the esophagus. *Med. Clin. M. Amer.*, 1927, *10*, 919–925.
7. Wile, Udo J. Syphilis of the esophagus. *Am. J. M. Sc.*, 1914, *148*, 180–186.

PYELO-URETERITIS CYSTICA*

WITH REPORT OF A CASE

By WALTER D. BIEBERBACH, M.D., *Urologist*, PHILIP H. COOK, M.D., *Radiologist*, *and* RAYMOND H. GOODALE, M.D., *Pathologist*
Worcester City Hospital
WORCESTER, MASSACHUSETTS

URETERITIS cystica is a well-recognized disease when seen at operation or autopsy, but a preoperative diagnosis is seldom correctly made. This may be accounted for by the fact that diagnosis has to be made from the patient's history and symptoms, combined with laboratory findings. These findings lead more often to a diagnosis of ureteral calculi, or disease of the kidney. The disease has been known for a long time and was first described by Morgagni, and illustrated in an Atlas by Rayer which was published in Paris in 1837. A disease known for so long a time and so seldom reported makes us suspicious that many such cases pass unrecognized through the hands of physicians and clinics. In the past such facilities as the roentgen ray and cystoscopy were not available. With our present equipment a diagnosis should be more readily made, providing the physician has had the opportunity of studying a case with correlation of these methods with the clinical findings.

To illustrate these remarks we shall analyze a case which was observed and operated upon on the urological service of the Worcester City Hospital. In the report and discussion of this case we would like to bring out the importance of intravenous and retrograde pyelography since, even with the advantages of cystoscopy and the roentgen ray, the average physician can fail in his preoperative diagnosis. This may be explained by the fact that many physicians pass through a lifetime of practice without seeing a case of this type and very few such cases are found in hospital records. When a case has been studied and observed, especially as to cystoscopic and roentgenologic findings, the appearance is very characteristic. It is fortunate that in reporting this particular case we have a complete check on the roentgen findings with both the intravenous and retrograde methods. This, we believe, is the only case reported with the combined findings, and in reviewing our hospital records it is the only case that has been recognized in this institution. This case had already passed through our hospital unrecognized, a diagnosis having been made of kink of the upper ureter and question of ureteral calculi. Such a diagnosis is reasonable when we realize that very few physicians have had the opportunity of seeing or studying ureteritis cystica and still fewer have ever seen intravenous or retrograde pyelograms of a disease of this type. It is our hope in reporting this case with the aid of our illustrations that more conditions of this kind will be recognized before operation.

REPORT OF CASE

The patient was a female, married, aged forty-two, born in Poland and a housewife by occupation. She had two admissions to the hospital, the first in November, 1931, at which time she came for pain in the left flank. The pain was noticed first three years before and was felt in the left kidney region. Three days after the onset of pain there was an attack of hematuria. The severe pain subsided on the appearance of blood in the urine and became duller in character accompanied by headache. She had no other symptoms. Gradually the pain and bleeding subsided and the patient would be free from symptoms for about two or three months. Lately they have recurred more frequently and the severity of the attacks and hemorrhage has increased. At the last attack, just before admission, the bleeding was not so severe as in former attacks, but the pain

* From the Departments of Urology, Roentgenology and Pathology, Worcester City Hospital.

was somewhat more severe. She had been seen by many physicians without results. Her history showed ordinary diseases of childhood with no complications and apparently good recoveries. She had no other illness except occasional temporal headaches. Family history negative for cancer and tuberculosis. Her general physical examination was negative except for a slight tenderness on deep palpation at the left costovertebral angle; no mass could be felt. The impression on admission was renal calculus. She was cystoscoped and examined roentgenologically, and the following report was made by the roentgenologist: "Kidneys outlined, normal in size and position. Catheters reach level of third transverse process. No calculi were seen. Bilateral ureteropyelograms show very small calyces on the right with no filling defects. The ureter is dilated to a maximum width of 1 cm. above the sacroiliac level. The lower portion is not well shown. On the left side pelvis and calyces within normal limits. The ureter shows an apparent right-angled kink with a hiatus amounting to nearly 1 cm. just above the third transverse process. There is very little dilatation. The ureter below the sacroiliac level is not well shown. This examination suggests partial obstruction of the ureters on both sides, on the right near the sacroiliac level and on the left in the region of the kink. The cause is not apparent and non-opaque calculus must be considered." The general cystoscopic findings in the bladder were relatively negative except for some congestion of the bladder mucosa. No apparent obstruction was felt to No. 7 roentgen catheters as they were passed to the pelvis of both kidneys. Bladder specimen of urine showed a slight trace of albumin, no sugar, rare red blood cells, frequent white blood cells, squamous epithelial cells and bacteria. Culture—non-pathogenic Gram-negative bacilli. Right kidney sediment rare red blood cells, occasional white blood cells. Culture—non-pathogenic, Gram-negative baccilli. A dye had been injected intravenously and appeared on both sides in twenty minutes. Blood chemistry: sugar 120 mg. per 100 c.c., non-protein nitrogen 30.0 mg. per 100 c.c.

The patient remained in the hospital three days and was discharged with the following notes: "Partial obstruction of left ureter due to kink, question of calculus." Her symptoms had improved following admission. It was difficult to obtain the full symptomatology since the patient could not speak English and an interpreter had to be used in questioning her. She was advised to return for further check-up if the symptoms returned. Figure 1 shows the findings at the first examination.

On January 31, 1933, she was readmitted to the urological service for further study and was examined by one of us (W. D. B.). Her symptoms were essentially the same as on her first admission but increased in severity, especially over the region of the left ureter, lasting from fifteen minutes to an entire day. There was now a constant dull ache in the left flank.

Fig. 1. Roentgenogram taken in 1931. Note ureteral kink with hiatus, and single vacuole, the significance of which was not realized. (In this and succeeding films the vacuoles have been slightly retouched for better reproduction.)

She had no burning or frequency on urination, but did notice that her urine was more bloody in appearance. She stated that following her last cystoscopy she was free from symptoms for two months. Her physical examination was essentially the same as on her former admission, with the chief outstanding symptoms, blood in the urine and tenderness over the left costovertebral angle. At no time while in the hospital was there a marked hematuria, but at times a catheter specimen would show many red blood cells in the sediment. Repeated routine urinal-

FIG. 2. Retrograde pyelogram made in 1933. Note increased ureteral dilatation and multiple vacuoles in left ureter.

yses were acid in reaction, with a specific gravity ranging from 1.010 to 1.015, an occasional trace of albumin but no sugar. Sediment showed occasionally a few white blood cells and many red blood cells. On one occasion report was made of the finding of many clumped white blood cells.

On the day of her admission her blood report was as follows: red blood cells, 4,600,000; white blood cells, 6,150; hemoglobin, 90 per cent. Smear—red blood cells show moderate variation in size and shape, no young forms, no achromia. Platelets reduced in number. Differential count; polymorphonuclears 55 per cent, lymphocytes 36 per cent, mononuclears 8 per cent, basophiles 2 per cent. Wassermann negative. Blood chemistry: alkali reserve 56.0 volumes per cent, sugar 100 mg. per 100 c.c., non-protein nitrogen 35.5 mg., creatinine 1.5 mg. per 100 c.c. Blood pressure was within normal limits. Temperature was normal except on third day when it reached 100°F. in the evening; it returned to normal in the morning and remained so. The costovertebral pain remained constant with increased ureteral colic on the left side and on February 5, 1933, she was cystoscoped with the following findings: No residual urine. Bladder capacity 300 c.c. Appearance—diffuse chronically inflamed bladder. Roentgen catheter No. 7 passed to the pelvis of each kidney and clear urine was collected from both sides with the following findings: Right kidney sediment showed occasional white blood cells and eipthelial cells. Culture—no growth. Left kidney sediment showed few white blood cells and epithelial cells. Culture—no growth. Bladder—negative for albumin and sugar; sediment showed few epithelial cells. Culture—no growth. Intravenous indigo-carmine appeared in good color from each side in four minutes. Plain roentgenograms and pyelograms both in the Trendelenburg and Fowler positions, 12.5 per cent sodium iodide being used for injection. The left side held 15 c.c. of fluid, the right 9 c.c.

The roentgen report was made as follows by the Department of Roentgenology: "Examination practically identical with that made in November, 1931, both ureters being dilated above the sacroiliac level. The left ureter shows numerous small vacuoles a few millimeters in diameter which may represent air bubbles. No calculi are shown in the tract." Figure 2 shows bilateral ureteropyelograms which are

FIG. 3. Intravenous pyelogram. Confirms previous observation by showing two vacuoles in left ureter.

similar to those taken on the first admission and the peculiar shadows can be seen in the left upper ureter and pelvis with the same kink that was seen before. Following this examination the ureteral colic symptoms disappeared but the constant dull ache remained in the left costovertebral angle and on February 13, nine days after the cystoscopy, intravenous pyelography was done and reported as follows: "Intravenous pyelograms show two vacuoles at level of the third transverse process. This indicates a pathological condition in the ureter. Diagnosis: Papilloma or similar growth." Figure 3 shows findings by intravenous ureteropyelography and we would like to call attention to the close resemblance to the retrograde method, thus showing the advisability of checking on questionable cases.

Fig. 4. Drawing showing cysts of kidney, pelvis and ureter.

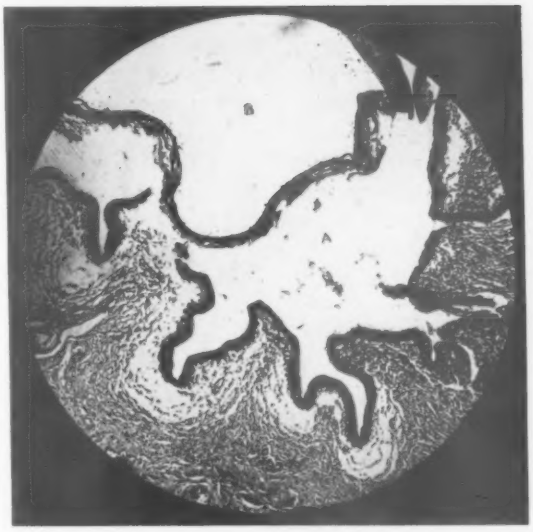


Fig. 5. Cross section of ureter. A, lumen of ureter; B, cyst projecting into lumen.

On February 14 it was decided that exploratory operation was indicated under ether anesthesia. The left kidney and upper ureter were explored by oblique costovertebral incision. As parts were delivered a distinct fixed kink was found 4 cm. below the ureteropelvic junction. The kidney was normal in size and soft. On palpation with the fingers just below the kink in the ureter, beady masses could be felt which extended down below the brim of the pelvis. By means of a small incision the ureter was opened for inspection and multiple growths of papillary appearance were observed and ureteronephrectomy was decided upon. The kidney was freed, the ureter clamped, tied and severed, renal pedicle clamped and tied and kidney removed. The oblique incision was closed in layers with drainage at the upper angle, the tied suture on the distal end of the ureter protruding at the lower end of the incision for traction. The patient was turned over on the back and a low rectus incision made, the peritoneum exposed and rolled inward, exposing the lower ureter which was freed and readily recognized by traction on the upper suture. This was ligated at the ureterovesical junction, thus removing the entire structure. The wound was closed with drainage in the lower angle. The removed specimen showed multiple small bluish cysts which studded the ureter and renal pelvis. The patient made an uneventful recovery, was out of bed on the fourteenth day and

left the hospital on the twentieth day, free from all symptoms. The blood chemistry on discharge was as follows: Sugar 120 mg. per 100 c.c., non-protein nitrogen 30.0 mg. per 100 c.c. Urinalysis was negative.

Pathological Report. Gross description: (1) A kidney measuring 9×6×4.5 cm. with a piece of ureter 5 cm. in length attached. The pelvis is not dilated. (2) A separate piece of ureter 12 cm. in length and averaging 0.8 cm. in diameter. The pelvis of the kidney and the opened ureter are covered with translucent elevations 2–3 mm. in diameter. These elevations are cystic and contain clear colorless fluid. They cover the entire mucosa of the pelvis of the kidney and the ureter (Fig. 4). Microscopic description: Ureter—a cross section of the ureter shows numerous folds of the mucosa with a round cell reaction immediately beneath it. The above-mentioned translucent elevations consist of cysts lined with a layer of cuboidal epithelium (Fig. 5). In the musculature are numerous foci of polymorphonuclear cells. Similar cysts are present in the pelvis of the kidney. Diagnosis: Pyelo-ureteritis cystica.

Since Morgagni[5] first described ureteritis cystica in 1761 there have been several cases reported but most of them were found at autopsy.

The only other report of roentgen diagnosis of this condition which we have found is the case of Joelson.[4] The patient was a female, aged forty-four, who complained of severe pain in the right lumbar region one week before admission. This was associated with gross hematuria. The cystoscopy revealed a cystitis cystica, and the ureterograms showed ureteritis cystica by retrograde pyelography. Calculi were found in both kidneys. No operation was reported.

The remaining cases were found at autopsy. Cleland's[2] report was that of a male, aged sixty-five, with bilateral ureteritis cystica. The pelvis of one kidney only contained the cysts. A case reported by Carson[1] was a female, aged fifty-eight, in whom the bladder wall, both ureters, left kidney pelvis, and the lower pelvis of the right kidney were covered with cysts varying in size from 1 to 3 mm. The right kidney pelvis was double, and the upper pelvis was not involved. Urquhart[7] reported 6 autopsied cases of coincidental ureteritis cystica and bilharziasis. He believes that the cystic condition was not caused by the presence of the bilharzia infection. Ages and sexes were not given.

Morse[6] studied 125 consecutive autopsies and found 3 instances of ureteritis cystica. None of these had urinary symptoms before death.

Etiology. Virchow[8] in a case report suggested that the cysts might be caused by the blocking of crypts in the mucosa of the ureter. Von Brunn's[9] theory, however, is most generally accepted. He believes that inflammation of the urinary tract causes a downward proliferation of the epithelium resulting in small groups of cells under the mucous membrane. Degeneration of the central cells of these buds results in the formation of cysts.

Giani[3] has been able to produce cystitis cystica in rabbits by the irritation of a foreign body in the bladder and by curetting the vesical mucosa through a suprapubic cystostomy.

DISCUSSION

Roentgen Diagnosis. The first examination, in 1931, showed a vaguely outlined filling defect a few millimeters in diameter in the contrast medium of the left ureter near the lower level of the third vertebra. With the possibility of non-opaque calculus as the cause of the symptoms, the roentgenologist allowed this to fit into the picture and considered no other conditions. In 1933, however, the vacuoles were numerous and well defined. Their multiplicity, their regular outline, and the fact that none of them appeared sufficiently large to cause the evident ureteral dilatation, argued against calculi. Air bubbles were next considered, but two of the lesions were seen in the same position at an intravenous pyelography several days after the first examination, which excluded this condition. Multiplicity and regular outline also seemed to exclude blood clots. The

only possible conclusion was that the vacuoles represented sessile tumors projecting into the lumen of the ureter, papillomatous or cystic in character. Later investigation of the few reported cases of papillomatous ureter show the roentgen appearances to differ somewhat from this, so that the picture here presented may fairly be taken as characteristic of ureteritis cystica.

There was no precedent in the literature advising operation in a case such as this. The patient's symptoms persisted unabated after the first cystoscopy. It seemed, therefore, that operative treatment was the only method of procedure. At the time of operation the exact diagnosis was not known, but in the presence of multiple tumors in the ureter it seemed advisable to remove the ureter and kidney. We recognize the possibility of the occurrence of the same condition on the other side. The patient to date, five months after operation, is free from symptoms.*†

SUMMARY

A case of unilateral pyelo-ureteritis cystica is reported illustrating the roentgen diagnosis of this disease by retrograde and intravenous pyelography.

The involved kidney and ureter were removed with relief of pain and hematuria. This appears to be the first operated case of pyelo-ureteritis cystica.

* The patient is still, one year after operation, free from symptoms. Retrograde pyelography in January, 1934, showed the right ureter negative.

† Since this article was submitted for publication in June, 1933, we have noted the report of a similar case by Kindall.[4a]

REFERENCES

1. Carson, W. J. Pyelitis, ureteritis and cystitis cystica. *Am. J. Surg.*, 1930, 8, 1256–1258.
2. Cleland, J. B. Ureteritis cystica. *M. J. Australia.* 1926, *2* 13.
3. Giani, R. Neuer experimenteller Beitrag zur Entstehung der Cystitis cystica. *Centralbl. f. allg. Path. u. path. Anat.*, 1906, *17*, 900–902. Also: *Beitr. z. path. Anat. u. z. allg. Path.*, 1907, *42*, 1–22.
4. Joelson, J. J. Pyelitis, ureteritis and cystitis cystica. *Arch. Surg.*, 1929, *18*, 1570–1583.
4a. Kindall, L. Pyelitis cystica and ureteritis cystica; report of case diagnosed by urography and confirmed by biopsy, with an outline of treatment. *J. Urol.*, 1933, *29*, 645.
5. Morgagni, J. B. The Seats and Causes of Diseases Investigated by Anatomy. Abridged and elucidated, with copious notes by William Cooke. Longman, London, 1822, Vol. II, pp. 316–319; 411–412.
6. Morse, H. D. Etiology and pathology of pyelitis cystica, ureteritis cystica and cystitis cystica. *Am. J. Path.*, 1928, *4*, 33–50.
7. Urquhart, A. L. Cyst formation in the ureter, associated with bilharziasis. *Brit. J. Urol.*, 1931, *3*, 21–25.
8. Virchow, R. Die krankhaften Geschwülste. August Hirschwald, Berlin, 1863, p. 246.
9. von Brunn, A. Ueber drüsenähnliche Bildungen in der Schleimhaut des Nierenbeckens der Ureters und der Harnblase beim Menchen. *Arch. f. mikr. Anat.*, 1893, *41*, 294–302.

ENCEPHALOGRAPHY UNDER NITROUS OXIDE ANESTHESIA*

By R. W. WAGGONER, M.D., D.Sc., *and* L. E. HIMLER, M.D.
ANN ARBOR, MICHIGAN

IT IS well known that the chief objection to encephalography is the reaction the patient experiences during and after the injection of air. Treatment of the post-encephalogram headache is of necessity symptomatic and usually presents no special difficulties. But while the procedure is taking place, no amount of sedatives short of stupefying doses will prevent the patient from being acutely uncomfortable. The severe headache complained of during this period often makes the patient resistive and not infrequently results in unsatisfactory or incomplete drainage. Ether anesthesia has been used for encephalography, but the danger of pulmonary complications and the unpleasant after-effects constitute distinct objections to this method. Solomon and Epstein[1] have reported a series of cases done under sodium ethyl barbiturate narcosis. The objection to this technique is that the narcosis is too profound and the effects too prolonged to render the method safe in all cases. This is especially true of children. We were led to do a series under nitrous oxide anesthesia in an attempt to find a middle ground between the disadvantages and discomforts attendant upon too light sedation and the dangers associated with extreme depression of consciousness while the air injection is being done.

METHOD

The procedure used in doing encephalograms under gas varies only slightly from that described by Camp and Waggoner.[2] While the anesthetic is given, the head of the chair is lowered to the prone position. A leather head-halter is used to keep the mask in place and it is fastened to the adjustable support on the back of the chair. When the patient is sufficiently anesthetized he is raised to the sitting position and the lumbar puncture needle is inserted in the usual way. The water manometer should be connected with sufficient length of small rubber tubing so it can be elevated to give pressure readings up to 1,000 mm., inasmuch as the standard two-piece manometer is only long enough to give a reading of 700 mm. above the puncture level. When the fluid stops draining freely, the head support is removed and the patient's head is rotated from side to side in order to complete drainage of the ventricles. The anesthetic is discontinued when no more fluid can be obtained, and the patient usually reacts within five minutes. Encephalograms are then taken immediately while the patient remains in the upright position.

COMMENTS

The greatest advantage of using gas is of course the fact that the patient is spared all subjective symptoms during the time the fluid is being removed. This means that the distressing headache and the apprehension which accompanies it at the beginning of the procedure are eliminated. If for any reason the operation must be interrupted suddenly, the gas can be discontinued and the patient reacts in a few minutes. This gives nitrous oxide a decided advantage over ether or a deep drug narcosis when emergencies arise. Toward the end of the procedure under gas the neck muscles are always relaxed well enough to permit free movement of the head in all directions. With no anesthetic this is often difficult, especially in uncooperative patients or those who develop an involuntary stiffness of the neck. When the patient comes to, his sensorium is still sufficiently depressed to obviate any serious discomfort, although he is quite capable of fairly

* From the Department of Neurology, University of Michigan, Ann Arbor.

TABLE SHOWING DETAILS OF
THIRTEEN REPRESENTATIVE ENCEPHALOGRAMS UNDER NITROUS OXIDE ANESTHESIA

Case	Sex	Age	Clinical Diagnosis	Encephalographic Findings	Time Draining (minutes)	Initial Pressure (mm. water)	Fluid Removed (c.c.)	W.B.C. Immediately Before	W.B.C. 24 hr. Later	Increase in W.B.C. Count
W.B.	M	3	Status epilepticus	Moderate internal hydrocephalus	45	590	180	7600	11,450	3850
J.N.	F	8	Jacksonian epilepsy	Essentially normal	60	490	65	5900	7,600	1700
R.V.	F	14	Epilepsy	Essentially normal	75	750	130	7950	12,350	4400
E.F.	F	15	Spastic tetraparesis	Internal hydrocephalus; bilateral cortical atrophy	50	750	130	8200	11,400	3200
J.R.	M	17	Behavior problem	Essentially normal	45	800	105	9850	11,900	2050
J.S.	M	18	Epilepsy	Extensive porencephaly	50	750	210	7200	10,000	2800
T.P.	M	20	Post-encephalitic Parkinsonism	Bilateral arachnoiditis over cortex	60	800	140	7600	14,050	6450
W.N.	M	23	? Traumatic encephalopathy	Arachnoiditis, cortical atrophy, congenital cyst	50	880	140	6800	8,950	2150
H.S.	M	24	Old skull fracture, epileptic attacks	Normal encephalogram	60	550	140	9250	15,050	5800
G.L.	M	24	Jacksonian attacks	Left cerebral lesion (operation: meningioma)	50	690	135	8000	11,300	3300
E.N.	M	30	Recent hemiplegia	No localizing evidence	45	700	100	9700	13,200	3500
E.K.	M	32	Jacksonian attacks	Right cerebral lesion (operation: astrocytoma)	40	750	120	6400	11,900	5500
A.K.	M	56	Traumatic encephalopathy	Bilateral cortical atrophy	65	850	155	6700	12,000	5300
Average Values					55	700	130			3900

complete cooperation during the film taking. It is our opinion that the use of nitrous oxide also tends to diminish the severity of the post-encephalographic reaction.

Pulse, blood pressure and respirations were recorded every few minutes in a series of cases, and no important variations in the condition of patients under nitrous oxide was revealed. In fact, without anesthesia the pulse frequently becomes weak and thready after about 50 c.c. of spinal fluid has been removed, while under gas

the pulse always remains full and firm, and the rate seldom exceeds 110-120 per minute. Stimulants have not been necessary in any of the first 50 cases studied under this method.

As the accompanying table indicates, encephalograms under gas have been done on patients ranging in age from two to fifty-six years. The average time required for the drainage of spinal fluid is slightly under one hour. The initial pressure reading when inhalation anesthetic is used varies between 450 and 900 mm., and remains moderately high, that is, above 500 mm., until well over half the total fluid is removed. After this it drops rather rapidly to zero. This elevated initial pressure has not led to any serious complications in our experience. As the chart shows, there is a fairly consistent rise in the leucocyte count within twenty-four hours after the encephalogram. This increase of from 2,000 to 6,000 white blood cells per cu. mm. is the same whether or not an anesthetic is used. Differential counts in a few cases have revealed no significant variations in the polymorphonuclear-lymphocyte ratio with either method.

No cases with choked discs or suspected posterior fossa tumors have been included thus far, although two with frontal and temporal tumors were clearly localized by this method. These two patients were up and free from headache in less than forty-eight hours. We were also able to study three other patients who have had two encephalograms, the first without and the second with gas, and each of them stated preference for the latter method.

CONCLUSIONS

In our experience with nitrous oxide anesthesia in encephalography we have found that this method decreases the patient's discomfort both during and after the procedure, without at the same time increasing the danger. After comparing its advantages and disadvantages with those of ether and non-volatile anesthesia, we believe that this is the method of choice in all but certain selected cases. The only objection to the use of nitrous oxide anesthesia in encephalography is the additional expense involved. The increase in initial intracranial pressure consequent upon its use does not appear to be a contraindication.

REFERENCES

1. SOLOMON, H. C., and EPSTEIN, S. H. Encephalography under narcosis produced by nonvolatile anesthetics. *J. Am. M. Ass.*, 1932, *98*, 1794–1796.
2. CAMP, C. D., and WAGGONER, R. W. The technic of encephalography. *Arch. Neurol. & Psychiat.*, 1931, *25*, 128–136.

Vol. XXXI, No. 6

CARCINOMA IN CHRONIC OSTEOMYELITIS

By J. J. COLLINS, M.D.
Roentgenologist, The John D. Archbold Memorial Hospital
THOMASVILLE, GEORGIA

A CASE of carcinoma, developing in a chronic osteomyelitic sinus of long standing, recently admitted to the John D. Archbold Memorial Hospital, was the stimulus for a review of the literature on this subject. A striking thing in this connection was the paucity of American literature on this condition and the very few recent publications, either in this country or abroad, in regard to it.

The condition is one that has long been known to occur. Cases were reported and discussed in the literature many years ago. Marjolin,[4] in 1828, described malignant changes in old ulcers. Following this, a number of cases of carcinoma occurring in chronic osteomyelitic sinuses were reported so that in 1894 Devars[2] was able to collect a series of 39 such cases from the literature. During the past twenty years, however, American publications along this line seem to be limited to those of Hitzrot,[3] Young,[7] and Benedict.[1] In the foreign literature, Vernengo,[6] in 1928, and Mathieu and Khan,[5] in 1920, published case reports.

Devars, in his series, found that the tibia was involved in 26 cases, the femur in 5, the bones of the foot in 5, the bones of the arm in 2 and the knee in one case. He also reported that the inguinal glands were often involved, a statement that is not substantiated by the cases reported more recently.

Vernengo reported a case occurring in a young man, aged twenty-two, whose history of osteomyelitis of the tibia dated back nine years. This is apparently the earliest age at which the condition is reported to have occurred. The treatment was amputation.

Young, in the "Cabot Case Records," discussed a case which was reported in a man, aged fifty-eight, who had a history of chronic osteomyelitis of the femur with a sinus of twenty years' duration. The preoperative diagnosis was chronic osteomyelitis only. After operation, the microscopic examination of the material removed from the bone cavity proved it to be epidermoid carcinoma. The inguinal glands were thought to be involved but proved to be inflammatory so that disarticulation at the hip was done.

Benedict, in a survey of 2,400 cases of osteomyelitis treated in the wards of the Massachusetts General Hospital, found 12 cases in which epidermoid carcinoma developed in chronic osteomyelitic sinuses. Males only were affected in this series and the areas of occurrence were limited to the lower extremities; 3 cases involved the foot, one the femur and 8 cases the tibia. There was no inguinal gland involvement in any of the cases reported and no distant metastases.

An analysis of the data given in this report shows that practically all of the cases developing carcinoma had a history of very long standing bone disease with sinus formation. It is interesting to note, in this connection, that the majority of cases occurred in individuals past fifty years of age, the youngest being thirty-four and the oldest seventy-five. The duration of bone infection was, on the average, slightly more than thirty-five years. The shortest duration of sinus formation previous to malignant development was one year; the longest was sixty-five years.

The clinical findings were those common to chronic osteomyelitis with discharging sinuses but with the added features of extremely foul smelling discharge and, in the majority of cases, either a cauliflower mass at the ostium of the sinus or an epithelial hyperplasia suggestive of carcinoma. Definite or suggestive local clinical evidence of malignancy was present in 9 of

the 12 cases in this report. In one case the diagnosis was made only after a second biopsy and in 2 cases it was made postoperatively in clinically unsuspected cases.

The roentgen appearance was not typical or characteristic as in most cases of metastatic carcinoma of bone. This is probably due to the fact that the condition is superimposed on chronic osteomyelitic changes accumulated over a long period of years and it is difficult to differentiate an area of destruction due to active bone infection from one due to malignant involvement in these cases. Then, too, because of the origin of the carcinoma at the edge or within the sinus, there may be no involvement of bone in the earlier stages.

The treatment was primary amputation in 7 cases; amputation very soon after excision in 2 cases in which the diagnosis was made postoperatively; excision only in 2 cases; excision followed by amputation for recurrence four months later in one case. No recurrence following these surgical measures was reported in any of these cases and no distant metastases occurred in the cases that were followed up.

The incidence of carcinoma in a series of 2,400 cases of osteomyelitis, as analyzed above, is 0.5 per cent. Considering the Cohnheim theory, that irritation is a cause of cancer, it is rather surprising that the incidence of carcinomatous change is so low in infections of bone which, as a class, are notoriously chronic. The irritation of continuous discharge from sinuses, the trauma of daily dressings, frequent surgical intervention, healing, temporary in character, with subsequent breaking down, a constant attempt at epithelial repair, would all seem to provide an ideal condition for the development of cancer from the irritation standpoint.

Definite conclusions as to incidence cannot, however, be based on the analysis of one large series of osteomyelitic cases. It is of course necessary to know what percentage of these are chronic cases as carcinomatous changes are not expected to occur in those that recover promptly after the acute stage. It is also necessary to know that a thorough search has been made for microscopic evidence of malignancy in all chronic cases treated. A bacteriological examination is much more apt to be made in chronic osteomyelitis than a search for microscopic evidence of malignancy of the tissue in and about a discharging sinus. If the diagnosis is not made in the early stages, the patient may eventually return with a typical cauliflower growth but it is possible that a certain percentage of these with early changes may be cured by resection or excision under the diagnosis of chronic osteomyelitis and for this reason are never diagnosed as carcinomatous. This is a possibility in this type of cancer as the malignancy of it seems to be of low grade and metastasis or glandular involvement very unusual.

To date, 43 cases of chronic osteomyelitis have been admitted to the John D. Archbold Memorial Hospital. Of this number, 2 cases have been diagnosed as carcinoma developing in a chronic osteomyelitic sinus of the tibia.

CASE 1. The patient was a white male, aged forty-three, who, at the age of four years, had developed an acute osteomyelitis of the tibia. Since that time he has had a sinus over the midtibial region anteriorly which intermittently healed and broke down. Six years ago, a granulating ulcer appeared about the mouth of the sinus and gradually increased in size. Because of this ulcer he came to the hospital in August, 1927, for treatment.

Examination at that time revealed an ulceration of the skin and soft tissues over the entire lateral and anterior aspects of the middle third of the tibia and fibula. Roentgen examination revealed a marked thickening and sclerosis of the tibia and fibula with several small areas of bone destruction in the middle third of the tibia. The diagnosis was chronic osteomyelitis. The operative report: Excision of ulcerated area of the skin and soft tissues. The anterior portion of the cortex of the middle third of the tibia was removed and several small abscess pockets were curetted. No microscopic examination of the tissue removed was made. Three weeks later a skin graft was done to cover the

Fig. 1. Case 1. Anterior view of leg showing large cauliflower mass with extensive scar above mass.

Fig. 2. Case 1. Lateral view of leg.

Fig. 3. Case 1. Roentgenogram of tibia and fibula showing areas of bone destruction and marked sclerosis of the bones.

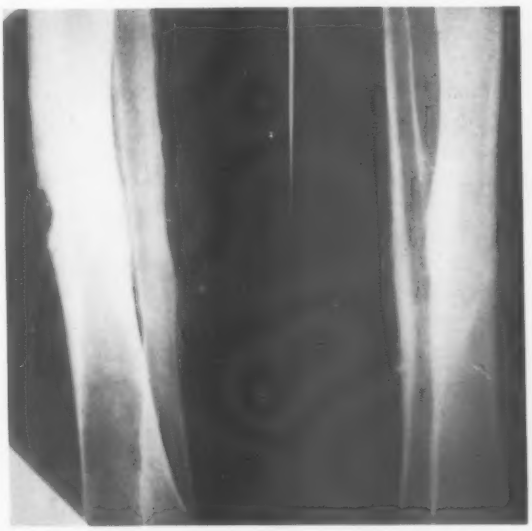


Fig. 4. Case 11. Roentgenogram of tibia and fibula showing periostitis of the fibula, sclerosis of both bones in mid-portion and area of bone destruction of the tibia.

large denuded area. The skin graft in the region of the old sinus did not take. The patient was discharged from the hospital much relieved as to pain but still had a discharging sinus.

The history subsequent to this is interesting. The wound healed well except for a small area about the sinus opening. Here, according to the patient, "proud flesh" soon appeared. He did not consult a doctor but burned this down with glacial acetic acid. It reappeared and, periodically, for the next five years, he burned this down with acid. In August, 1931, the growth became so rapid that chemical cautery was no longer effective and a large cauliflower mass developed. In December, 1931, he again returned to the hospital.

At this time there was a very large cauliflower mass on the anterior aspect of the middle third of the left tibial region. In the middle of the mass was a sinus which discharged extremely foul smelling pus. The tumor tissue bled profusely with any manipulation. There was no apparent involvement of the glands of the groin. Films of the chest showed no metastases. The roentgen examination of the left tibia and fibula showed a marked sclerosis of the tibia and fibula with a large area of bone destruction and sequestration in the middle third of the tibia. Biopsy was done and the microscopic pathological report was squamous cell carcinoma. Amputation was advised but refused. The patient wished to try roentgen and radium therapy. This was applied with no appreciable response. Consent to amputation was then obtained and the leg was removed in the mid-femoral region.

Examination of the amputated leg revealed an extension of the carcinoma into the bone with a large cavity in the tibia filled with an extremely foul smelling cheesy material.

In reviewing the record of the previous admission of this patient to the hospital, it is notable that a large ulcerated area about a chronic sinus was present. The ulcerated area was excised but no microscopic examination of the tissue was made. It is reasonable to suppose, in view of these findings and in the light of the subsequent course, that carcinoma was present at least in the soft tissues at the time of the operation in August, 1927. The fact that its growth was held in check over a five year period by chemical cauterization, the apparent absence of glandular involvement or distant metastases speaks for a very low grade of malignancy.*

CASE II. M. M. J. (No. 8265), admitted to the John D. Archbold Memorial Hospital on September 24, 1929. Chief complaint: Ulcer on the right leg, anterior aspect, midway between the ankle and knee.

Present trouble began about one year ago after a severe contusion over the anterior tibia received in an automobile accident. On the day following the accident, the patient had a severe chill followed by fever, pain in the leg below the knee and difficulty in moving the leg. A short time later a sinus developed over the mid-tibial area anteriorly and has persisted since then. Several months ago, ulceration about the sinus was noted. This has gradually increased in size and for past few weeks has bled profusely at times.

Examination of the right leg: On the anterior surface, in the middle third of the tibia, there is an ulcer the size of one's hand. The edges are irregular and the ulcer itself is composed of granular masses 1 to 2 cm. in diameter. There are no enlarged veins in the leg. No enlarged glands in the inguinal region.

The Wassermann reaction was negative. A biopsy of the ulcerated area proved it to be squamous cell carcinoma.

The roentgen examination showed a periostitis of the entire fibula with a rather marked sclerosis of the middle thirds of the tibia and fibula. There was an area of destruction of the sclerotic cortex of the tibia which had the appearance of having been "scooped out." The appearance suggested a malignant destruction of that area.

The treatment was amputation just below the knee. Operative recovery was uneventful. No follow-up record is available.

SUMMARY

Very little literature has appeared in recent years in regard to carcinoma developing in chronic osteomyelitic sinuses. The cases reported have, for the most part,

* In Case I the tissue removed at operation in August, 1927, has recently been examined microscopically and areas of epidermoid carcinoma found in it. Proved malignancy was thus present in this case for a period of five years previous to amputation of the leg. The patient has now been alive and well for two years following amputation.

occurred in very long-standing bone infections. The diagnosis may or may not be apparent clinically. The condition is of low malignancy, regional gland involvement and metastasis apparently being rare. The prognosis with adequate excision or amputation seems to be good. The roentgen findings are not typical or suggestive of malignancy in most cases.

The incidence of this condition seems very low but it is possible that bearing the condition in mind and making a microscopic examination of excised tissue and chronic sinus tracts will bring to light more cases than are now being diagnosed.

The ideal treatment of any disease is, of course, prevention of that disease. It is highly probable, however, that chronic osteomyelitic sinuses will continue to be seen in appreciable numbers and every effort should be made to diagnose malignant changes in these while they are in a sufficiently early stage to be treated by excision or resection rather than by amputation.

REFERENCES

1. Benedict, E. B. Carcinoma in osteomyelitis. *Surg., Gynec. & Ost.*, 1931, *53*, 1–11.
2. Devars, M. Quoted by Benedict, ref. 1.
3. Hitzrot, J. M. Epithelioma in sinus of old osteomyelitis. *Ann. Surg.*, 1921, *73*, 247.
4. Marjolin. Quoted by Benedict, ref. 1.
5. Mathieu, P., and Kahn, T. Quoted by Benedict, ref. 1.
6. Vernengo, M. J. Epitelioma desarrollado en una fistula por osteomielitis crónica. *Bol. y trab. d e la Soc. de cir. de Buenos Aires*, 1928, *12*, 801–810.
7. Young, E. L., Jr. Cabot Case Records. *New England J. M.*, 1930, *203*, 373.

THE SOLUTION OF THE ROENTGEN DIAGNOSTIC PROBLEM IN CHRONIC APPENDICITIS

CONCLUSIONS BASED UPON STUDIES COVERING A PERIOD OF TWENTY YEARS AND INCLUDING A COMPARATIVE ANALYSIS OF ROENTGENOLOGICAL, CLINICAL, SURGICAL AND POST-MORTEM FINDINGS

By THOMAS SCHOLZ, M.D.
NEW YORK CITY

IF STATISTICS are correct in their conclusions that the mortality from appendicitis is on the increase, while at the same time approximately 40 per cent of all appendectomies are done unnecessarily, there must be something wrong with the diagnosis of this disease.

In considering the diagnostic means in appendicitis our attention is automatically first attracted by roentgenology. A method which can visualize the appendix or at least definitely determine its location, which, therefore, under the fluoroscopic screen can act as a guide for the examiner's palpating hand, and which evidently also may be used as a differential diagnostic means, ought to be an ideal diagnostic aid.

Indeed, after the introduction of the contrast meal method, attempts at using the roentgen ray for the diagnosis of appendicitis were made early. Bennett,[3] Holzknecht,[18] and Béclère[2] were the first to discuss this subject.

Bennett came to the conclusion that, though the roentgenograms often show nothing, they may occasionally be of inestimable value, especially in differential diagnostic respect, "provided a competent radiographer is available." Holzknecht also stressed the importance of the roentgen rays for the purpose of differential diagnosis. Béclère considered the roentgen method of special value for the surgeon by showing him the position of the appendix, and for the clinician by determining whether or not a tender point corresponds to the appendix.

With the technical improvements beginning about 1910 and resulting in a more successful visualization of the various parts of the alimentary canal and a better roentgenological understanding of the digestive system, roentgen diagnostic efforts in appendicitis received a new stimulus.

Studies were made as to the demonstrability of the appendix and the diagnostic value of the direct information thus obtained regarding this organ. Attention was also paid to combinations of such direct roentgen evidence with indirect signs observed either in the appendix itself or in adjacent or more remote parts. Thus, up to the present time, there have been declared by various writers the following roentgen signs, occurring singly or in various combinations, as more or less characteristic or suggestive of chronic appendicitis: demonstrability and non-demonstrability of the appendix; abnormalities as to its location (abnormally high or low, retrocecal, retro-ileal, etc.); abnormalities as to its size and shape (abnormally long, kinked, coiled up, segmented, etc.); adhesions; spasm; retention; concretions; local tenderness; ileocecal stasis; insufficiency of the ileocecal valve; cecal stasis; gastric retention; spastic phenomena in cecum, colon, stomach and duodenum, etc.

However, in regard to the significance and diagnostic value of these various roentgen signs no accord of opinion could yet be reached. On the contrary, opinions in this respect differ so greatly that in many instances they are even diametrically opposite. To illustrate this controversy a few examples may briefly be mentioned.

Rappaport (cited by Gottheiner[16]) considers every demonstrable appendix as displeased, while Czepa,[12] Hüffer and Kaspar,[19] and Buisson[6] declare non-demonstra-

bility of the appendix the most important sign of pathology.

In regard to frequency of visualization, Case[8] demonstrated the appendix in 30 per cent of the cases, Hurst[20] in 50 per cent, George and Gerber[15] in 70 per cent and Moore and Merritt[29] in 80 per cent.

Buisson claims to be able to visualize every normal appendix. According to Gottheiner[16] the appendix can be shown in the vast majority of instances if the observations are made often enough, or by repeating the examination in a week or two. Application of catharsis, he says, may through increased peristalsis cause the filling of an otherwise non-demonstrable appendix. Cohn[10] proved that the appendix may fill and empty several times in the course of an examination. In order to be useful the observations must therefore be made frequently enough. Henszelmann[17] and Orndoff[30] recommend special technical arrangements for the purpose of a clearer visualization of the appendix and the cecal region.

Singer and Holzknecht,[36] on the other hand, warn against these efforts to demonstrate the organ. They consider them useless, because visualization of the cecum alone, they say, is sufficient for diagnostic conclusions. Gottheiner, also, believed that special technical arrangements are unnecessary and thinks that intensive observation coupled with experience is the best guide.

A number of writers, among them especially Singer and Holzknecht,[36] Pfahler,[31] Imboden,[21] Carman,[7] Childs,[9] White,[41] Koerbler,[25] Moore and Merritt,[29] and Jacquet and Gally,[22] lay greatest stress upon local tenderness.

Holzknecht thinks that local tenderness is the only reliable and important sign, for the determination of which visualization of the cecum alone is sufficient. Imboden believes local tenderness to be of paramount importance. To elicit it properly he recommends roentgenoscopy with the patient in recumbent posture, and careful palpation. For better visualization of the appendix in cases of ptosis and better recognition of adhesions he advises the Trendelenburg position. Carman is of the opinion that without local tenderness "all the other signs lose much of their persuasiveness." Childs says that "a tender appendix roentgenologically demonstrated carries far more clinical value than tenderness over McBurney's point." White considers local tenderness the best sign of pathology and adds that "a patient rarely needs operation if there is no tenderness when the appendix is moved under the palpating finger."

Gottheiner, Buisson, and others, on the other hand, disagree with the above views. Gottheiner especially criticizes Holzknecht for his attitude regarding local tenderness. Though it may be granted, he says, that local tenderness is of importance, there are other equally or even more important signs.

Retention in the appendix is considered as definitely pathological by many writers, especially George and Gerber,[15] while Carman[7] considers it of problematical value, and Assmann[1] has definitely proved that it may occur in perfectly normal appendices.

The differential diagnostic aid offered by the roentgen ray is entirely ignored or only generally mentioned by some writers, while others lay great stress upon it or even consider it the most important part of the roentgen procedure.

Pfahler, for instance, states that he has seen several cases of renal calculi operated upon for appendicitis. By a proper utilization of the roentgen method one could avoid such mistakes. Gottheiner emphasizes that as a good clinician always examines the entire patient, so also a good roentgenologist should not only pay attention to the appendix, but should go over the entire digestive tract, including the intrathoracic organs; only then will he be able to exclude the possibility of other lesions being responsible for the patient's symptoms. He considers roentgenoscopy alone sufficient for such purposes and criticizes the American roentgenologists for taking too many plates. Most outspoken in regard to the importance of the differ-

ential diagnosis, however, is Carman when he says: "When by the roentgen examination the stomach and duodenum may reasonably be excluded, the X-ray has given its most valuable service and should not as yet be expected to go much further."

As to the examination procedure itself, extremely few examiners systematically take abdominal plates before application of the contrast meal followed by a complete gastrointestinal series. The latter is begun either in the usual manner or the Haudek meal method is used. Some examiners begin the actual observations immediately after application of the barium meal, others at the six hour or nine hour period or even later. Some observers rely solely upon roentgenoscopy, others use roentgenoscopy and plates.

Thus it is seen that up to the present time roentgenologic studies have failed to develop in appendicitis any definite method of procedure or any definite and reliable roentgenographic sign-complex such as for instance has been worked out and universally accepted in gastric and duodenal ulcer, gastric carcinoma and other digestive disturbances. On the contrary, there is no other abdominal condition in regard to the roentgen diagnosis of which there still exists as much difference of opinion as in appendicitis. This controversy goes so far that while many roentgenologists, as we have seen, claim to be able to make a more or less definite diagnosis in chronic appendicitis, many clinicians consider such a diagnosis as unreliable and even call it "a delusion and a snare."

Why this controversy of opinion? Does the cause for it lie in the method itself, or its wrong application and utilization? Or are there possibly some other disturbing factors the elimination of which we have not yet learned?

These questions aroused my interest early in my roentgenologic career, and have finally prompted me to undertake certain systematic studies for the purpose of possibly solving them. As it is felt that these studies have at last succeeded in producing results sufficient for definite conclusions, it was decided to make them the subject of this paper.

The roentgenological observations which gradually led up to this work were made, aside from two years of institutional roentgen experience in Germany, in the roentgen-ray department of Mt. Sinai Hospital during the years 1911 to 1914, while the special studies were conducted in the roentgen-ray departments of the Montefiore Home and the Lebanon Hospital during the years 1915 to 1920, and in private practice since.

For the sake of a better understanding of the points at issue it will be necessary to describe first, though very briefly, my preliminary observations in regard to the roentgen diagnosis of chronic appendicitis, because it is that practical experience which revealed the main questions upon the solution of which a firm foundation for the roentgen diagnosis would have to depend. Furthermore, it is the analysis of that first experience which suggested the best possible methods for the solution of those questions.

Afterwards the arrangement of the various investigation methods will be described, then their results will be discussed, and finally the conclusions will be drawn.

In the consideration of the subject, discussion will be confined to chronic appendicitis as in acute appendicitis roentgen examinations are, as a rule, contraindicated.

Preliminary Observation

Observations regarding the roentgen diagnosis in appendicitis began on a larger scale in the roentgen-ray department of the Mt. Sinai hospital in 1911 and were continued there until 1914. The hospital proper and a busy out-patient department furnished sufficiently large material. Full time work, without the interference of private practice, gave opportunity for a certain amount of comparative studies of roentgenological, clinical and surgical find-

ings. The clinical understanding of obscure cases was greatly helped by an ever ready cooperation on the part of the hospital visiting staff for which due appreciation is herewith offered.

The greatest aid, however, came from an ideal teacher. In this connection it is not only a special pleasure, but a duty to express most sincere thanks to Dr. Leopold Jaches, Director of the roentgen department of the Mt. Sinai Hospital, for his untiring efforts in properly guiding, during those years, my roentgenological development. His seasoned and conservative judgment, healthful restraining and warning against undue diagnostic overenthusiasm greatly facilitated the evaluation of the various roentgen signs and thus laid a foundation without which this work would have been impossible.

My personal impressions gained on the basis of these preliminary observations may briefly be summed up thus:

The appendix was visualized, without any special technical arrangements, in a vast majority of cases. Occasionally, an appendix not demonstrated at the first examination became visible at a later reexamination if such was necessary for some other reason.

A retrocecal appendix not seen with the cecum completely filled, became recognizable with the cecum partly emptied, or with the aid of a luffa sponge placed underneath the patient, or with the patient in oblique posture. In cases of extreme ptosis, visualization of the appendix sometimes met with great difficulty. In most of these instances, however, one succeeded by means of pressure manipulations or a contrast enema in lifting up the cecal region sufficiently to allow a satisfactory inspection of the pericecal region.

The so-called roentgen signs enumerated in the introductory paragraph were observed in various frequencies. Local tenderness, however, as elicited by palpation under the roentgenoscope, seemed to be the most constant and most reliable diagnostic evidence. Less constant and regular in occurrence appeared to be direct findings as to the appendix. Still more variable were indirect signs such as spastic phenomena, stagnation, etc.

Not infrequently the diagnostic problem was greatly complicated by apparently coexisting lesions, like gastric or duodenal ulcer, gallstones, urinary calculi, etc. Where such findings were not associated with appendicular roentgen signs, the diagnosis appeared simple and the differential diagnostic value of the roentgen ray was impressive. In instances, however, where these lesions were associated with appendical roentgen findings the diagnostic situation sometimes became extremely puzzling for the inexperienced and even taxed the ability of the experienced examiner.

In comparing the direct roentgen findings with the situation revealed by surgical exploration, it was found that the roentgenographic information regarding the location of the appendix was always correct so that the surgeons gradually seemed to appreciate the value of such preoperative knowledge. Other roentgenographic evidences were usually, but not uniformly, confirmed. There were instances where adhesions, kinking, concretions, etc., were either not found at operation or had been overlooked at the examination.

The checking-up of the correctness of the roentgen diagnosis was done by the usual method of comparison with the anatomical diagnosis furnished by the surgeon or the pathologist. However, these results finally became somewhat puzzling for more than one reason.

Where surgical inspection and macroscopic investigation revealed definite evidence of pathology, for instance in form of pus, or gangrene, intramural abscesses, erosions of the mucosa, thickening and induration of the appendix wall, infiltration of peri-appendicular tissues, etc., the anatomical diagnosis seemed to be settled beyond any doubt. But there were cases in which macroscopic investigation showed a

perfectly normal appendix so that the latter had to be subjected to microscopic examination. And in every one of these latter instances an anatomical diagnosis of appendicitis was rendered.

Furthermore, there were encountered in the roentgen department not infrequently patients in whom a comparatively short time ago the appendix had been removed, either in this or some other hospital, with evidently completely negative therapeutic results. An investigation revealed that in practically all these cases, too, a diagnosis of appendicitis had been made roentgenologically and anatomically.

The subsequent roentgen examination revealed in a vast majority of these latter cases definite evidence of various important lesions which, on clinical analysis, most probably had existed at the time of the previous roentgen examination and had been the cause of the symptoms, and, on roentgenological analysis, probably should have been detected by the roentgenologist at that time. This, together with the negative therapeutic results, aroused the suspicion that the original clinical, roentgenological and anatomical diagnoses might have been wrong, that the operation might have been unnecessary, and that the roentgenologist might possibly have been able to prevent this apparently unnecessary operation.

It therefore seemed that the result of a check-up of the roentgen diagnosis would depend upon what one chose as the standard for the comparison. If the roentgen findings were compared with the anatomical diagnosis, the roentgen diagnosis evidently was 100 per cent correct; on the other hand, if the roentgen diagnosis had been checked with the therapeutic results, many diagnostic errors would evidently have had to be registered. Therefore, also, if one had cared at that time to rush into print one could have, according to the selection of the standard of comparison, proved or disproved the diagnostic validity of practically any of the various roentgen signs of appendicitis.

Problem to be Solved as Indicated by these Preliminary Observations

An analysis of the above experiences indicated that the solution of the roentgen diagnostic problem in appendicitis would depend upon a further investigation of the following three questions:

1. Is local tenderness, as ascertained roentgenoscopically, really the only reliable sign of appendicitis? And what is the diagnostic status of the other so-called roentgen signs?
2. What rôle may roentgenology play in differential diagnostic respect? To what extent may it reliably serve as a means for preventing unnecessary operations? And what is the best roentgenological procedure for such purpose?
3. Are anatomical findings or therapeutic results the best criteria for the correctness of the diagnosis of chronic appendicitis?

Best Basis for the Solution of the Problem

A detailed study of all the phases of the problem seemed to indicate that a further investigation in this direction would have the best chance for satisfactory results if it were arranged along the following lines.

The question of the diagnostic value of local tenderness could probably be settled by making, in a sufficiently large follow-up series of cases, such tenderness alone, as elicited by palpation under the roentgenoscope, the deciding diagnostic factor. This conclusion was arrived at on the strength of the following reasoning: First, previous experience seemed to indicate that out of the great multitude of assumed roentgen signs local tenderness was the most constant and most reliable one. Secondly, if the clinical diagnosis of chronic appendicitis was based, at least in adults, outside of history, almost exclusively upon tenderness elicited by the clinician's palpating hand over the assumed location of the non-visualized appendix, then local tenderness elicited by the roentgenologist's

finger over the definitely localized appendix should with much more justification be taken as the deciding diagnostic finding. Thirdly, many of the other roentgen signs, especially those giving direct information as to the appearance of the appendix, could not very well be used for diagnostic conclusions, because a small, easily flexible and movable organ as the appendix, having besides, a very narrow lumen, would naturally be expected to present a great variability in its roentgenographic appearance even under normal conditions.

The real diagnostic status of all the various roentgen signs other than local tenderness could best be ascertained by recording the frequency of their occurrence in appendicitis and non-appendicitis cases.

In regard to the verification of the roentgen diagnosis it was evident that, in view of the uniformly positive pathological findings in the removed appendices, one could not very well expect reliable information as to the correctness of the diagnosis from the usual method of comparing roentgenographic and anatomical findings. More reliable results in this respect probably would be obtained if, in addition to the anatomic control, one would also follow up the therapeutic effect in the operated patients.

The differential diagnostic question could probably be decided by systematically paying attention in every appendix case to all those roentgenographically demonstrable lesions which possibly may enter into the causation of the patient's symptoms. Additional information would be obtained by a systematic roentgenological analysis of appendectomy cases with negative therapeutic results. Such observations, if carried on over a long period of time, would enable one, it was hoped, to work out a definite roentgenological procedure by means of which the differential diagnostic points could be exhaustively covered as far as the roentgenologist was concerned.

The question of the true status of the anatomical changes as a criterion for the correctness of the roentgen diagnosis could best be solved by a post-mortem study of previously roentgenographed clinically normal appendices. A study of this type ought to enable one to determine what pathological changes, if any, may exist in such appendices; to what extent, if at all, anatomical changes may take place in an appendix without giving rise to clinical symptoms; and to what degree, if at all, such changes may participate in the causation of the various appendicular roentgen signs.

Opportunity for Such Research

It probably was a fortunate coincidence that an unusual opportunity for carrying on this type of investigation offered itself in the following years in the roentgen-ray departments of the Montefiore Home and the Lebanon Hospital.

It was the combination of the work in these two institutions which proved so fortunate. While the Lebanon Hospital with its acute service furnished sufficient material for practical routine observations in appendicitis cases, without burdening the department with any constant rush of work, the Montefiore Home with its chronic material gave an excellent opportunity for prolonged roentgenologic observations of clinically normal appendices. And as a large percentage of these patients remained in the institution until their death, there was a possibility to compare, in a large number of instances, the previously obtained appendicular roentgen findings with the autopsy evidence.

The roentgen studies in the Montefiore Home entailed a considerable additional expense for the department. In fact, their result would have been greatly jeopardized, unless the hospital authorities, especially Dr. Siegfried Wachsmann, at that time Medical Director of the institution, had given the writer carte blanche in regard to such expenses. This generosity is herewith greatly appreciated.

The anatomical study of the appendices was greatly facilitated by an unusually cordial cooperation on the part of members of the pathological departments, especially

Dr. Eugene Bernstein. For all this help and also for aid coming from many other quarters most sincere thanks are herewith expressed.

After the investigations in the two hospitals had rendered tangible results and had cleared the investigator's vision regarding the problems in question, private practice until the present time gave an opportunity for studying in regard to the differential diagnostic question certain phases which usually are not met with in institutional roentgen-ray work.

Arrangement of the Material

After many unsuccessful attempts at the solution of the problem there gradually was evolved a definite system in the arrangement of the material and the method of procedure.

In order to assure uniformity of the results, I decided to do the entire work. Therefore only such cases were taken into consideration in which all the observations had been made by myself.

From the great mass of accumulated material the following series of investigations has been made the basis for my final conclusions:

1. A roentgenologic study of 100 hospital patients referred for examination of the appendix during the years 1915 to 1919.

2. A roentgenologic study of another series of 100 patients referred in private practice for examination of the appendix during the years 1926 to 1931.

This second series was decided upon in view of the progress made in the roentgenology of the digestive system since the first series, especially through the introduction of the gallbladder visualization examination.

3. A roentgenologic study of a series of 100 clinically normal appendices.

4. A comparative study of roentgenologic and post-mortem findings in 46 clinically normal appendices.

5. A roentgenologic analysis of 63 therapeutically negative appendectomy cases collected in private practice during the years 1921 until 1931.

The methods of procedure employed in these investigations will be described in the following.

1. Examination Series for Appendicitis

The examination procedure in this series was arranged along the lines previously mentioned, making local tenderness the deciding roentgen diagnostic factor, recording all other appendical roentgen findings, and paying attention to the differential diagnostic possibilities. To accomplish these aims it was decided, on the basis of previous experiences, to use the following:

Examination Method. Patients with constipation received a light physic the night before, and a plain cleansing enema on the morning of the examination. Otherwise the patient was given only the latter. The importance of the cleansing of the large intestine for the roentgen examination of the appendix will be referred to later.

Prior to the application of the contrast meal, plates of the entire urinary tract were taken in such a manner that the lumbar and sacral spine and both hip joints were included. These plates were developed immediately and studied. If necessary, additional confirmatory plates were made.

The contrast medium was given either in milk or zoolak, usually the latter. The examination was begun in the morning with the patient on empty stomach. The Haudek meal method could not very well be used unless the preliminary plates were taken on the previous day.

Always the entire digestive canal was examined, using roentgenoscopy and plates. During the roentgenoscopic observation special attention was paid to the intrathoracic organs for the exclusion of possible pulmonary and diaphragmatic lesions. In positive or doubtful instances chest plates were made.

The second observation was usually made after six hours, except where the stomach seemed to empty rapidly, when

it was done earlier. Further observations took place not in a routine fashion at definitely set periods, but were arranged individually according to existing motility conditions.

The examination was considered finished as soon as the colon, including the appendix, had emptied. In cases of stasis or apparent non-demonstrability of the appendix, the observations were continued until the seventy-two hour period, but no longer, because previous experience had shown that a further prolongation of the examination had only an academic, but no practical value.

For a better visualization of the cecum or appendix in cases of ptosis, elevation of the pelvis or the contrast enema method, in retrocecal appendices, compression by means of a luffa sponge or an oblique view were used. Otherwise no special technical efforts or apparatus were employed.

The diagnosis was made, as explained above, upon local tenderness alone as elicited by palpation under the guide of the roentgenoscope. Visualization of the appendix itself was not considered essential. Its localization by the visualized cecum was taken as sufficient for the purpose. All other appendical roentgen findings, which according to the literature had been designated as signs of chronic appendicitis, were also noted and recorded.

The determination of local tenderness was at times very difficult, especially during the first part of the series. However, with increasing experience in the technique of palpation and in the proper sizing up of the mental attitude of each individual patient, this difficulty greatly lessened.

The problem of properly evaluating appendicular tenderness in instances of multiple tender points was usually satisfactorily solved with the aid of the above experience or with the help of coexisting signs pointing in the direction of some other lesion. By extending the roentgenologic search in that direction, whether in the form of an additional gallbladder examination, or an additional examination by means of the barium enema for a possible colonic lesion, etc., the question could usually be solved.

A very valuable aid in this respect was derived from statements volunteered by the patient, so that later on it was made a rule to obtain in the course of the observations a complete history directly from the patient. It may be stated that without such "talks" with the patient a hidden lesion would occasionally have escaped roentgen detection at that time.

It was of course realized that an examination procedure as just outlined did not exclude all the differential diagnostic possibilities, because it was still necessary to keep in mind certain gynecological and urinary conditions which either cannot be detected at all by the roentgen ray or cannot be shown by a flat plate. However, it was felt that the method assured the maximum that could reasonably be expected from roentgenology in this type of case without interference on the part of the roentgenologist with the clinician's domain. In roentgenologically negative instances, the clinician would then have to decide on the strength of the clinical data, whether or not an additional roentgen investigation would be advisable.

The same examination procedure was used in the later series of 100 private patients except that the gallbladder visualization examination was used very extensively for differential diagnostic purposes.

The final diagnostic conclusions were drawn on a very conservative basis. Cases presenting either definite appendical tenderness alone or definite predominance of such tenderness as compared with other tender points were classified as appendicitis (Table 1). Cases showing definite evidence of some other important lesion were classified as such even if there was some appendical tenderness, because experience teaches that one very rarely makes a mistake by explaining, especially in abdominal conditions, all the patient's symptoms on the basis of one definitely diagnosed cause.

Patients who presented either no local tenderness or only indefinite tenderness or

Table I

ROENTGEN DIAGNOSES IN TWO SERIES, OF 100 CASES EACH, OF PATIENTS REFERRED FOR ROENTGEN EXAMINATON OF THE APPENDIX

Roentgen Diagnosis	In 100 Hospital Cases 1915–1919		In 100 Private Cases 1926–1931	
Appendicitis (on the basis of tender appendix)	45		37	
Negative	19		24	
Gallbladder lesion with stones	2		6	
Gallbladder lesion without stones	3		8	
Gastric ulcer	2		3	
Duodenal ulcer	8		5	
Extreme visceroptosis	6		4	
Cecum mobile	2		1	
Carcinoma of cecum	1		—	
Carcinoma of ascending colon	—	36	1	39
Carcinoma of sigmoid	1		2	
Advanced diverticulitis of sigmoid	1		3	
Meckel's diverticulum	1		—	
Right renal stone	4		2	
Right ureteral stone	2		1	
Movable right kidney	1		3	
Right lower lobe pneumonia	—		—	
Right diaphragmatic pleurisy	1		—	
Subphrenic abscess	1		—	
Advanced sacroiliac disease	—		1	

several questionable tender points without evident basis, or in whom the mental attitude suggested neurasthenia, etc., were classified as negative.

Series of 100 Clinically Normal Appendices

Patients for this series were selected on the basis of history, physical examination and roentgenological survey.

Only those cases were chosen which according to their history never had had anything that could be interpreted as an appendicitis attack; which presented no appendical tenderness, no evidence of digestive or urinary disturbance, no suggestion of pelvic trouble or disease of the lower spine or right hip; which showed no stigmata of neurasthenia or hysteria; and which had had no abdominal operations.

As in the previously mentioned two series, here, too, the entire digestive canal was roentgenographed and the examination was considered finished as soon as the colon including the appendix and cecum had emptied. In cases of cecal and appendical stasis, however, the observations were not discontinued at the seventy-two hour period as in the previous series, but were continued until the barium had disappeared from those parts, which sometimes took as long as eight to twelve days.

In the course of these observations special attention was paid to all those findings which had ever been designated as roentgen signs of chronic appendicitis. Such findings were recorded and later tabulated (Table II). The further course of these cases was then kept track of.

Post-Mortem Series of 46 Clinically Normal Appendices

Of the above series of 100 roentgenologically examined patients with clinically normal appendices, 59 died in the institution during the period of the investigation. In 46 of these 59 cases either the appendix or a complete autopsy was obtained.

The gross anatomical findings thus secured were compared with the roentgen evidence. Appendices which presented a normal macroscopic appearance were subjected to microscopic examination.

Series of 63 Appendectomy Cases with Negative Therapeutic Results

During the period of 1921 to 1931 there were collected in private practice 63 cases which had previously been diagnosed roentgenologically as appendicitis and had been operated upon with completely negative therapeutic results, and in whom the subsequent roentgen examination revealed evidence of some other important lesion as the probable cause of the patient's original symptoms.

The cases were collected on a very conservative basis. Only such cases were included in which the history definitely in-

TABLE II

ROENTGEN FINDINGS, SUPPOSED TO BE SUGGESTIVE OF APPENDICITIS, AS OBSERVED IN APPENDICITIS CASES, OTHER ABDOMINAL LESIONS, AND IN CLINICALLY NORMAL APPENDICES

Roentgen Findings	In 74 Appendicitis Cases	In 77 Other Abdominal Lesions	In 100 Clinically Normal Appendices
Appendix visualized	63	62	81
Appendix not visualized	11	15	19
Appendicular tenderness	74	8	—
Appendix abnormally high	7	4	6
Appendix abnormally low	8	11	9
Appendix abnormally long	3	5	2
Appendix displaced medianward	2	—	4
Appendix along external lateral aspect	1	3	2
Appendix retrocecal	5	3	6
Appendix retro-ileal	3	1	4
Appendix kinked	6	5	9
Appendix coiled up	3	1	4
Appendicular adhesions	13	10	18
Irregular appendicular outline (segmented, etc.)	12	15	21
Appendicular concretions	2	1	3
Ileocecal stasis 24 hr.+	2	4	3
Appendicular retention 72 hr.+	8	11	14
Cecal retention 48 hr.+	9	16	11
Gastric retention 6 hr.	3	9	5
Pyloroduodenal spasm	7	22	4
Colonic spasm	8	14	6

dicated that the present lesion in all probability already existed at the time of the roentgen examination for appendicitis, and where the further clinical course made it reasonably safe that the patient's symptoms at that time were due to the now discovered lesion. Cases where there was any doubt in this respect, and also those which had been operated upon without roentgen examination, were excluded. Otherwise the series would have been appreciably larger.

Results of above Investigations

The results of the series of investigations just described have been arranged for the purpose of clearness and brevity, as far as possible, in the form of tables. For the sake of convenience they will be discussed under the following four headings:

1. Roentgen diagnostic value of local tenderness and—
   Best criterion for the correctness of the diagnosis.
2. Evaluation of all other roentgen signs of appendicitis.
3. Differential diagnostic problem.
4. Post-mortem study of clinically normal appendices.

1. *Roentgen Diagnostic Value of Local Tenderness* and *Best Criterion for the Correctness of the Diagnosis*

In the first series of 100 patients, as is seen from Table 1, a definite diagnosis of appendicitis solely on the basis of local tenderness as elicited by palpation under the roentgenoscope was made in 45 cases. Of these 45 patients, 39 were operated upon.

Among the negative cases 2 suffered a typical appendicitis attack after two and five weeks respectively. One was re-examined and a diagnosis of appendicitis made on the strength of a characteristic local

tenderness. This case also came to operation.

In these 40 operated cases (with no mortality) the removed appendix was declared "pathological" in every instance. In 32 appendices the anatomical diagnosis was based upon macroscopic and in 8 upon microscopic findings.

On the basis of the anatomic findings the roentgen diagnosis, therefore, was 100 per cent correct.

Inquiries as to the therapeutic result in these 40 operated cases revealed that in 37 there was complete relief from the symptoms or very marked improvement, while in 3 instances the original symptoms persisted.

The further follow-up of these 3 non-improved cases showed that one, presenting stigmata of neurasthenia, continued going from one physician to another and finally was operated upon again, this time for "postoperative adhesions." In another one combined cystoscopic and roentgenographic examination revealed a kink of the right ureter at the level of the fourth lumbar vertebra. In the third a fibroid of the uterus was removed nine months later. Both of these latter patients were relieved of their symptoms by the respective treatments.

On the basis of the therapeutic results, therefore, the roentgen diagnosis was correct in only 92.5 per cent of the cases.

In the private series of 100 patients (Table 1) a diagnosis of appendicitis on the basis of roentgenoscopically determined local tenderness alone was made in 37 instances, followed by operation in 31 cases.

Among the 24 negative cases 4 showed within the next three to nine weeks reappearance of symptoms referable to the appendix region. Three of these 4 patients were re-examined and a diagnosis of appendicitis was made on the strength of local tenderness, and operation was performed.

In these 34 operated cases, also, the removed appendix was declared "pathological" in every instance. In 29 the anatomical diagnosis was made macroscopically, in 5 microscopically.

Here, too, therefore, the roentgen diagnosis as checked up by the anatomical findings was 100 per cent correct.

The follow-up of the therapeutic results, possible in 33 of the 34 cases, revealed that 29 patients considered themselves relieved of their previous symptoms, while in 4 the trouble persisted or has even become aggravated by the operation. In one of these 4 unrelieved cases a pus kidney was removed six months later; another one was operated upon for a right-sided hernia; and a third was being treated for "adnexitis," followed by improvement or complete cure in all. In the fourth patient the persistence of the symptoms could not be explained.

On the basis of the therapeutic results, therefore, in this series, too, the correctness of the roentgen diagnosis was appreciably lower than when checked up on the basis of the anatomic findings, namely, only 89 per cent.

To the above may be added that in 2 cases operated upon for a calculous cholecystitis, roentgenographically diagnosed, and in a case of chronic obstructive duodenal ulcer, in all of which coexistence of appendical tenderness had been observed roentgenoscopically, the simultaneously removed appendix was also declared "pathological" on microscopic examination. In another instance of roentgenologically diagnosed advanced gallbladder lesion, also associated with marked appendical tenderness, where the appendix was not removed the appendical tenderness was found entirely absent at a roentgenoscopic re-examination eleven months later.

These observations, therefore, justify in regard to (*a*) local tenderness and (*b*) criterion of diagnosis, the following definite conclusions:

(*a*) *Local tenderness as elicited by palpation over the roentgenoscopically visualized appendix or appendix region is a reliable diagnostic sign of appendicitis* because it enables one to make a definitely proved correct diagnosis in approximately 90 per cent of the cases.

Absence of appendical tenderness does not exclude the presence of an appendix lesion. It only means that at the time of the examination there was in the appendix no actual inflammatory process. It is therefore important that the roentgen examination be made during the period of symptoms or be repeated on the recurrence of the attack.

Occasionally appendicular tenderness on roentgenoscopic palpation may be simulated by some extra-appendicular lesion such as cannot be recognized roentgenologically. This phase of the problem will be discussed later.

(*b*) *The therapeutic result is a better criterion for the correctness of the diagnosis of chronic appendicitis than anatomical findings* because anatomically a diagnosis of appendicitis was made also in those instances where the appendectomy effected no relief whatever, where it was later proved that the symptoms were due to some other cause, and where treatment for such cause brought about a complete cure.

This statement, however, has to be modified to the effect that in those instances where surgical inspection and macroscopic investigation revealed unmistakable evidence of inflammation the operation always proved a therapeutic success.

2. *Evaluation of Roentgen Signs other than Local Tenderness*

For the evaluation of all so-called roentgen signs of chronic appendicitis, other than local tenderness, reference should be made to Table II. In this table is found analyzed the frequency of occurrence of such signs as observed in 74 appendicitis cases, 77 instances of other abdominal lesions, and 100 clinically normal appendices.

A study of the table shows that the incidence of these "roentgen signs of appendicitis" presents no characteristic preponderance in definitely proved appendicitis cases as compared with that in other abdominal conditions or in clinically normal appendices. On the contrary, some of these signs, like cecal and appendical stasis and spastic phenomena, occur more frequently in non-appendicitis cases.

It must be concluded, therefore, that outside of local tenderness, *none of the directly or indirectly obtained roentgen evidences, which hitherto have been considered signs of appendicitis, have any diagnostic value.*

Especially should be pointed out the diagnostic uselessness of appendical retention which in some of the clinically normal appendices persisted for eight to twelve days, because not infrequently even now a definite diagnosis of appendicitis is being made and operation is being advised on this sign alone.

The roentgenologist, furthermore, should be most urgently warned against the indiscriminate diagnostic use of spastic phenomena, the variability of which has been pointed out by us on a previous occasion.[14] As useful as their occurrence in the upper parts of the digestive canal often is for the diagnosis of various lesions, as useless diagnostically is it in the lower parts. In fact, most of such so-called spastic phenomena in the appendix itself and in the colon are not the product of appendicitis, but of immature experience.

In regard to the anatomical findings in cases presenting these various appendical roentgen signs, the reader is referred to the post-mortem study of clinically normal appendices.

3. *Differential Diagnostic Problem*

The magnitude of the differential diagnostic problem in appendicitis is shown in Table I which contains an analysis of the diagnostic situation in the two series, of 100 patients each, previously described.

In justice to the clinician it must be stated that in these 200 patients referred for roentgen examination of the appendix, the clinical diagnosis was not always appendicitis. In many instances the required roentgen examination was intended merely for the purpose of exclusion, while the real clinical diagnosis not infrequently coincided with the subsequent roentgen findings.

However, Table 1 reveals which roentgenologically definitely demonstrable conditions may give rise to symptoms simulating the clinical picture of appendicitis. In this regard the summary at once impresses upon us the large number and the great variety of such lesions and the consequently important rôle roentgenology is called upon to play in differential diagnostic respect.

Although the roentgenographic recognition of these various differential diagnostic lesions is a matter of common knowledge among roentgenologists, a few points of special importance may be emphasized.

It is of interest to note that, while in the series before introduction of cholecystography duodenal ulcer was the most frequent differential diagnostic lesion, after its introduction gallbladder disease has assumed the first place. Cholecystography therefore should be very extensively used for differential diagnostic purposes.

In gastric and duodenal ulcers, and especially in gallbladder lesions, not infrequently coexisting appendical tenderness is observed. The association of this finding with the above lesions is of great interest and importance because in some instances the appendicular tenderness may clinically as well as roentgenoscopically so completely overshadow the other lesion that both the clinician and the roentgenologist may be induced to arrive at a diagnosis of appendicitis. The watchful roentgenologist can correct such misleading impression by definitely proving the existence of the other lesion.

Of course, one may argue that in such instances the appendicitis is the primary and more important, and the ulcer or gallbladder condition the secondary lesion. This argument seems to be supported by Kruchen[26] who reported cases of appendicitis with typical symptoms of gastric ulcer and by Moynihan's statement that "most ulcers are found in the appendix." However, one should take into consideration the fact that the roentgen diagnosis of ulcer, and for that matter of most other digestive disturbances, is not based upon an evidence like clinical symptoms, the interpretation of which under the most favorable conditions is correct in approximately only one-third of the cases, but upon data in the interpretation of which the experienced roentgenologist is correct in more than 90 per cent. This together with the further results of our investigation will disprove Moynihan's statement, at least as far as its application to roentgenology may be concerned.

When finding definite evidence of ulcer or gallbladder lesion the roentgenologist, therefore, will do well to lay more stress upon such lesions than upon a coexisting appendical tenderness. This seems to be also corroborated by the previously mentioned gallbladder case in which the tenderness over the appendix cleared up after the removal of the gallbladder alone. It would lead too far here to try to explain these phenomena on the basis of lymph or nerve supply or some other theory.

Of special differential diagnostic interest are cecum mobile and extreme visceroptosis because, here, owing to the usually present cecal tenderness, the differentiation is very difficult, and because an unnecessary appendectomy on account of the frequency of subsequent adhesions usually markedly aggravates the original symptoms. Though

TABLE III

ROENTGEN FINDINGS IN 63 PATIENTS, PREVIOUSLY OPERATED ON FOR APPENDICITIS, WITH NEGATIVE THERAPEUTIC RESULTS

Gallbladder lesions	with stones	8
	without stones	12
Duodenal ulcer		13
Gastric ulcer		5
Gastric carcinoma		3
Right renal stone		6
Right ureteral stone		9
Advanced diverticulitis of sigmoid		3
Carcinoma of sigmoid		2
Carcinoma of 5th lumbar vertebra (Ca. of lung)		1
Carcinoma right sacroiliac region (Ca. of breast)		1

through the work of Wilms,[43] Stierlin [39] and Blake[5] the importance of the mobile cecum is now generally recognized, the roentgenologist should be reminded that its rôle in the causation of appendicitis has been greatly overestimated. It is now well known, especially through the investigations of Aschoff, that, exclusive of tuberculosis, inflammatory conditions very rarely spread from the cecum into the appendix. In the presence of a cecum mobile one should therefore hesitate to make a roentgen diagnosis of appendicitis unless there is exceptionally definite evidence.

The real value of the roentgen ray as a differential diagnostic means, however, is realized when looking over Table III the contents of which have been collected in the manner before described. As a detailed analysis of these cases will be given in a separate paper, it may suffice here to point out only a few roentgenological features.

The question naturally asked regarding this material is: How was it possible that the lesions enumerated in this table, so definitely demonstrable by the roentgen ray, could be so completely overlooked at the roentgen examination of the appendix? As the answer to this question was considered of extreme importance for our subject all efforts were bent upon obtaining as much information as possible regarding the character of the previous roentgen examination and the type of the examiner.

In view of the fact, however, that the roentgen observations (and to a certain extent also the operations) in these cases had been made in four different countries and in five different states of this country, the information obtained was not as complete as was desired. In only 51 instances was it possible to gain sufficient data in form of the original roentgen report, or the plates, or information supplied by the previous physician. In the remaining 12 cases one had to rely in this respect solely upon the patient. Yet by making due allowance for evidently incorrect statements on the part of the patient, the information thus gained gives a fairly correct picture of the reasons for the roentgen diagnostic mistake.

These inquiries revealed the following information as to the character of the roentgen examination:

Entire Gastrointestinal Tract		Intestinal Tract Only		Examination without Barium Meal	Plates before Barium Meal Followed by Complete Gastrointestinal Series
24		35			
Roentgenoscopy and Plates	Roentgenoscopy Alone	Roentgenoscopy and Plates	Roentgenoscopy Alone		
18	6	23	12	4	None

As to the various types of roentgen examiners and the number of cases examined by each, the following information was obtained:

Competent Roentgenologists	Incompetent Medical Roentgen Examiners		Technicians Working for Roentgenologist or Clinician or Their Own Account
	44		
	Various "X-ray Laboratories"	Clinicians Doing Their Own X-ray Work	
5	19	25	14

The above analysis explains the reasons for the diagnostic mistakes so clearly that no further discussion is necessary.

It is easily seen that a *faulty technical procedure* has caused the errors. Some of the lesions were overlooked because only the lower half of the digestive canal was examined; others, because roentgenoscopy

alone was used; still others, because no special plates of the urinary tract and spine were taken, etc. That a roentgen examination for appendicitis without the application of a contrast medium may not be quite satisfactory is also clear.

The most common single factor primarily instrumental in the causation of this faulty technique with its consequent errors, was *incompetence on the part of the roentgen examiner*. And it was extremely painful to observe that clinicians, who should be the first in taking care of the patient's interests, were the most frequent offenders in this respect by either doing the work themselves or leaving it entirely to their nurses, or by referring their patients to incompetent men. And it was still more painful to note that among such clinicians were men of international renown.

The harm done by these differential diagnostic errors was in many instances very great because the vast majority of these patients belonged to the middle class. When seeing how some of them had spent their entire savings for surgeon and hospital on the promise that they would quickly become well again; how others had been thrown into debt or even had been financially ruined; how in still other instances the happiness of the entire family had been destroyed; and, finally, how some of the patients had in the meanwhile lost their last chance for recovery, one could not help having a deep feeling of sympathy and shame.

It was therefore not at all surprising to note that practically all these patients or their family members expressed doubt as to the honesty of the physicians. Most of them openly showed dislike and contempt for the medical profession and came for the new roentgen examination only reluctantly and with distrust.

From the above we must conclude that, owing to the great multitude of serious, reontgenologically definitely demonstrable lesions which may simulate the clinical picture of appendicitis, and owing to the great harm that may result for the patient from the non-recognition of such lesions, the roentgen differential diagnosis is by far more important than the roentgen diagnosis of appendicitis itself.

To do the patient full justice in this respect calls for a systematic roentgen survey will all the differential diagnostic possibilities in mind. It must always cover the entire gastrointestinal tract, chest, urinary tract and lower spine. If necessary, special examinations of gallbladder, colon, etc., must be added.

On account of its diversity and difficulty such a roentgen survey can be successfully made only by an experienced and competent roentgenologist.

It must here again be added that certain urinary lesions, neuropathic conditions like neurasthenia and hysteria, or gynecological diseases, may even then upset the roentgen diagnosis. A careful history will in such instances be the saving factor.

The differential diagnostic importance of urinary conditions, other than those demonstrable by a flat plate, has been shown by Lowsley and Twinem.[28] Special examinations with the aid of uroselectan or the combined cystoscopic and roentgenographic method may therefore occasionally be called for.

Some of the differential diagnostic lesions, for instance carcinoma of the lower spine, may to some of us appear extremely rare and therefore of little practical value. Yet I have previously[34, 35] shown that such conditions, if secondary to a clinically unrecognized or unrecognizable primary focus in the chest, thyroid, breast, etc., may just on account of their rareness and obscure nature lead to very grave diagnostic errors which by a proper utilization of the roentgen method are easily avoidable.

That Gottheiner's statement that roentgenoscopy alone is sufficient for differential diagnostic purposes is absolutely wrong is self-evident.

4. *Post-Mortem Study in 46 Clinically Normal Appendices*

This study comprises (*a*) a macroscopic investigation, (*b*) a microscopic examina-

tion of the macroscopically normal appendices, and (*c*) a comparison of the gross anatomical condition with the intra vitam roentgen findings.

(*a*) *Macroscopic Anatomical Findings.* The cross anatomical condition was decided by inspection and cross sections. As pathological were considered only changes in the appendix itself or in the periappendicular tissues. Adhesions, kinking, etc., were not classified as pathologic, but as findings the pathological significance of which had yet to be ascertained.

Definitely recognizable gross pathology in the appendix itself was observed in 8 cases. These changes, occurring singly or in various combinations, consisted of a marked localized thickening of the subserous and intramuscular connective tissue, or very pronounced induration and thickening of the entire appendix, sometimes associated with stenosis or even complete obliteration of the lumen, small evidently healed defects in the wall, healed erosions in the mucosa, brown discoloration, etc. In one instance the appendix was rudimentary and hardly recognizable.

In no case, however, was there any sign of actual inflammation like free pus, exudate, fibrinous covering of the serosa, intramural abscesses, etc.

Periappendicular changes were found in 2 cases, in one of them in association with appendical findings.

Chronic gross pathology, therefore, was noted in 10 cases (21 per cent), while the remaining 36 appendices presented a normal macroscopic appearance.

Conclusions from the Above. Chronic anatomical changes, even of the most advanced type, may occur without any symptoms or signs and even without a history of previous appendicitis attacks. In other words, even most pronounced chronic anatomical changes in the appendix may be merely an accidental finding having nothing to do with the causation of the patient's symptoms.

This confirms a similar observation by Williams and Slater[42] and the opinion of Connell[11] who doubted if what are called chronic anatomic changes in the appendix are really always the cause of the patient's symptoms.

(*b*) *Microscopic Findings.* Extremely illuminating were the microscopic findings in the 36 macroscopically normal appendices.

All but 2 presented evidence of anatomical changes. These changes usually consisted of a more or less pronounced increase in the subserous and intramuscular connective tissue or of round cell infiltration of the submucosa.

The 2 normal appendices were in younger persons, and as one of them was associated with a marked thickening and sclerosis of the periappendicular tissues, there was really only one completely normal appendix.

However, the value of these microscopic findings became somewhat doubtful on account of the following observation made during this investigation. When looking over and studying with the pathologist a large number of the slides the impression was gained that there seems to exist a great uncertainty in regard to what is normal and what is pathological. During such arguments the pathologist was often unable to decide definitely where the normal ends and the abnormal begins. And when showing some of these slides to different pathologists different opinions were obtained.

Conclusions from the Above. If microscopic anatomical changes are looked for, they will be found in practically every appendix, especially in adults. In regard to the nature and the significance of these changes there exists a marked difference of opinion among pathologists. However, as they occurred here in clinically normal appendices, it must be definitely assumed that microscopic anatomic findings of the type described above have no pathological significance.

(*c*) *Comparative Study of Gross Anatomical and Roentgenological Findings.* An analysis of this comparison is presented in Table IV. In this table the macroscopic changes in the appendix itself are entered

TABLE IV

COMPARISON OF ROENTGEN AND POST-MORTEM FINDINGS IN 46 CLINICALLY NORMAL APPENDICES

Roentgen findings occurring singly or in various combinations		Gross pathological findings in appendix and adjacent tissues
Non-visualized appendices	9	Appendix greatly thickened with lumen completely obliterated in 2; stenosed at base 1; kinked by adhesions 1; rudimentary appendix 1; normal appendix and lumen in 4.
37 roentgenographically visualized appendices		
Appendix irregularly outlined (segmented, etc.)	11	Mural changes, interfering with lumen in 3; not interfering with lumen in 1; normal appendix and lumen in 7.
Appendix retrocecal	3 with adhesions	Position confirmed; adhesions in 2; mural changes in 1; normal appendix in 2.
Appendix retro-ileal	1	Postion confirmed, no adhesions; normal appendix.
Appendix along external lateral cecal aspect	1 with adhesions	Position confirmed; no adhesions; normal appendix.
Appendix kinked	3 with adhesions	Kinked, with adhesions and mural changes, in 1; normal appendix in 2.
Appendix coiled up	1 with adhesions	Coiled up, no adhesions, normal appendix.
Appendix abnormally long	2	Abnormally long; no mural changes.
Appendix abnormally high	3 with adhesions in 2	Position confirmed; adhesions in 1; normal appendices.
Appendix abnormally low	5 with adhesions in 1	Position confirmed; adhesions in 2; mural changes in 1; normal appendix in 4.
Appendix displaced medianward	2	Position confirmed; no adhesions; normal appendices.
Retention in appendix 72 hr.	6 with adhesions in 1	Adhesions in 2; mural changes in 1; normal appendix in 5.
Retention in cecum 48 hr.	4	Pericecal and appendicular adhesions in 2; mural changes in 1; normal appendix in 3.
Ileocecal stasis 24 hr.	2	Adhesions in 1; normal appendices.
Appendicular concretions	2	Confirmed in 1, with mural changes; found in one other, with normal appendix.
Cases with adhesions	11	Confirmed in 8; not diagnosed in 3.

as "mural changes" in contradistinction to periappendicular findings, while the appendices with negative macroscopic observations are called "normal appendices." The discussion which follows is arranged according to the "roentgen signs of appendicitis."

Non-demonstrability of the Appendix. Among the 9 non-visualized appendices there was in 5 a definite anatomical basis, in the form of stenosis or obliteration, for their non-demonstrability, while in 4 the appendix and its lumen were perfectly normal.

For the non-visualization of these 4 normal appendices no definite reason can be offered. However, it may be more than a mere coincidence that 3 of these patients had been suffering from chronic constipation. Clogging of the appendix lumen by stagnating feces may therefore be a not far-fetched explanation. It was this possibility which suggested the advisability of having the patient's intestine cleansed before the beginning of the examination.

As the appendices presenting obliteration were observed in elderly people and as the anatomic changes producing it had not given rise to any symptoms or signs, this would tend to corroborate Skinner's[37] conclusion that such obliteration after thirty years is a purely physiologic phenomenon.

Irregular Appendical Outline. For the so commonly observed roentgenographic irregularities in the outline of the appendix, like segmentation, beading, etc., no anatomic explanation was found. It is true that in 3 instances there were anatomical changes in the wall encroaching in some places upon the appendical lumen, but not to the extent or in the shape suggested by the plate. In the vast majority of these instances, however, there was no anatomic reason for it whatsoever.

White,[41] therefore, was right in assuming that such irregularities have no pathological significance. Most probably they are produced by folds of the mucosa which, in view of the narrow lumen, may in places completely displace the contrast medium temporarily. This appears corroborated by the roentgenological observation that on different plates of the same appendix these irregularities appear on different levels, in different forms, or are present on one plate and absent on the other.

Appendical Adhesions. The post-mortem findings in regard to these adhesions confirmed the surgically proved fact that their occurrence is more common than the roentgen examination is able to reveal, but that in the majority of instances they can be diagnosed correctly. However, as they are found in clinically and gross anatomically normal appendices their significance is of no diagnostic value.

Our investigation also reminded us of the well-known fact that it is sometimes very difficult to determine periappendicular adhesions. As the appendix varies greatly in its relation to the peritoneal covering, sometimes being almost entirely extra-, another time completely intra-peritoneal, these varying peritoneal attachments at times simulate adhesions to such a degree that a differentiation becomes practically impossible.

Appendical Kinks. In this respect interesting information was obtained. Among the 3 cases of sharp kinking (not the commonly observed round bends) there was an anatomical explanation for the finding, in form of adhesion, in only one, while in the two others the appendix was slightly bent, but perfectly free and macroscopically normal.

Investigation of the peritoneal covering of these two appendices, however, revealed that the outer edge of the peritoneal attachments coincided with the level of the bends. Furthermore, it was found that by injecting fluid into these appendices a more or less sharp kink could be produced.

It is therefore evident that the outer edge of the peritoneal covering causes a weak point in the appendix wall so it may be possible that in such instances a normal bend might become intensified into a real kink by the weight of the contrast medium in the appendix.

Appendical Retention. In the vast majority of appendical stasis no anatomical explanation was found for the stagnation. It may occur in macroscopically perfectly normal appendices.

In view of this, new interest must be paid to the investigations of Waller and Cole[40] who have demonstrated in the appendix a definite sphincteric and peristaltic function. It would be of value to determine what factors, if any, may influence such function.

Ileocecal Stasis. An anatomical explanation for this condition, in the form of adhesions, was found in one instance, while in the other case there were no recognizable changes.

Abnormal Location. In regard to the great variation in the location of the appendix, an observation which had early attracted the attention of roentgenological investigators and more recently has again been carefully discussed by Peirce,[32] Kantor[23] and Kantor and Schechter,[24] the autopsies proved, as surgical findings have done before, that the roentgenographic information was correct in every instance. But they also revealed that no such deviation from the normal has any pathological significance. Such abnormalities, like retrocecal or retroileal appendix, etc., may occur in macroscopically perfectly normal organs.

Conclusions from this Comparative Study. Gross anatomical changes in the appendix, even if they are of the most advanced type, do not manifest themselves roentgenographically in any conclusively characteristic manner. In other words, from the roentgen appearance of the appendix we cannot draw any conclusions whatsoever as to its anatomical condition.

Comment

The presented material explains the great chaos that has existed up to the present in regard to the diagnosis of chronic appendicitis.

This was due to the fact that on the basis of an indiscriminate use of macroscopic and microscopic anatomical findings practically every removed appendix was declared diseased.

This again brought about a hardly believable carelessness in clinical and roentgenographic diagnosis of this condition and a disregard of the importance of the differential diagnosis, because no matter on what symptoms the clinician based his diagnosis the operation always confirmed his findings. Similarly in roentgenology, no matter what signs he chose, on what a small number of cases he based his conclusions, or how immature his experience was, the roentgen examiner could always bring proof, in the form of anatomic changes, for his diagnostic ability and the diagnostic value of his favorite roentgen sign.

One need not, of course, accuse the pathologist of any lack of good faith in rendering his diagnosis, because anatomic changes are really present in practically every appendix. What we are justified in criticizing, however, is his evaluation of such changes, an evaluation which obviously and very urgently calls for explanation and correction.

For instance, why is it that at autopsies not infrequently most advanced connective tissue changes, sometimes so pronounced as to cause complete obliteration of the appendix lumen, are explained to us on the basis of a purely regressive process of no pathological significance, while the same pathologist, when called upon to examine a removed appendix, will interpret the slightest connective tissue changes as evidence of inflammation? Furthermore, the question of the significance of the increase in the leucocytes in the mucosa and of their penetration through the epithelial layer, and other uncertainties, revealed in the course of our investigation, as to where the normal ends and the pathologic begins, await a solution on the part of the pathologist. Until such time we must decline to accept anatomical changes as a reliable criterion for the correctness of the diagnosis, but must rely in this respect rather upon the therapeutic result, in some in-

stances in conjunction with self-evident signs of inflammation as obtained by surgical inspection.

As we have proved that the roentgen diagnosis of appendicitis must be based upon one single sign, namely, roentgenoscopically determined local tenderness, with all other roentgen evidence having no diagnostic significance, it follows that the evidence furnished by the roentgenogram, no matter how long we may study it, cannot be made the basis of the diagnosis.

However, once the diagnosis is made roentgenoscopically then the lifeless plate becomes animated and begins to assume importance also by transmitting to the surgeon valuable advance information. To know that the appendix is situated abnormally high; or is hidden behind the cecum or ileum; or buried way down in the true pelvis; or situated in the left side of the abdomen; or associated with adhesive deformities of the cecum or other adjacent parts of the intestinal canal, etc., will to him be highly welcome preoperative news. And if such a plate is taken with the patient in recumbent posture and the rays coming from below so that displacements from pressure are avoided and the plate portrays the topographic conditions as they are found on the operating table, then the surgeon will still more consider the roentgenogram of real assistance.

We should therefore remember that local tenderness is the soul of the positive roentgen diagnosis of appendicitis. It instills life and significance into any other informative roentgen evidence. Without it all such other evidence assumes a purely incidental character.

Why it is that Deaver dislikes the roentgen examination in appendicitis especially "since the roentgen report stresses the presence of tenderness in the manipulation in carrying out the examination," while at the same time he greatly emphasizes the importance of such tenderness "over the location of the appendix during the physical examination," is not quite clear except that his statement may imply that the roentgenologist does not know how to palpate. It is true that proper palpation is an art that can be acquired only by practice and training. But this prerequisite applies to clinician, surgeon and roentgenologist alike. And there is no apparent reason for assuming that the latter lacks the intelligence or opportunity for acquiring this ability to as perfect a degree as anybody else.

Of especial diagnostic aid is roentgenoscopy in appendicitis in children, because here more than in adults symptoms are apt to be extremely puzzling. In a fretful and irritable child, or in one showing irregular appetite or bowel movement, or in one presenting loss of weight and ambition, without any apparent cause and with negative physical findings, a carefully done roentgenoscopic survey with light palpation over the easily visible and often displaced appendix region may quickly solve the diagnostic problem.

However, the real value of the roentgen examination in appendicitis lies, as we have seen, in the aid it offers in differential diagnosis. It is here that the roentgenologist by arranging his examination procedure so that all the roentgenographic differential diagnostic possibilities are covered, may reap diagnostic triumphs which cannot be equalled by any other examiner, no matter who he may be, provided that the roentgenologist is competent.

The necessity of experience on the part of the examiner must be especially stressed in private practice, because, while the hospital patient as a rule is excellently taken care of roentgenologically, it is the private patient who is most likely to fall into incompetent hands with a consequence of diagnostic errors. And it is mainly in private practice, where the vast majority of the patients consist of persons of moderate or very small means, that diagnostic errors as analyzed in Table III may exert such a disastrous effect.

The contents of this table do not exaggerate the situation. On the contrary, as the series comprises patients who prior to

the appendectomy had gone through the hands of all types of physicians, specialists and non-specialists, some of them having enjoyed preoperative advice by the highest medical and surgical authorities, here and abroad, it is fair to assume that the table gives a correct cross section of the differential diagnostic conditions in appendicitis as they prevail in private practice today.

The frequency of these diagnostic mistakes and their far reaching detrimental effect is realized neither by the practitioner, because the dissatisfied patient usually changes doctors, nor by roentgenologists, because we as a rule do our work quite blindly along the lines suggested by the referring clinician. It is only when in our private practice we become interested in the humane side of our work, when we care to acquaint ourselves with the history and story of these cases and begin to collect them, that the true state of affairs begins to dawn upon us. It is then that we begin to realize how cases, as shown in Table III, may greatly contribute to the growing animosity among the public against the medical profession and how they may also bring this specialty into undeserved disrepute.

In criticizing the clinician's roentgen activities we do not blame him for having and using a roentgen machine in his office for diagnostic purposes, as long as he knows the limitations of his roentgen diagnostic ability. Roentgen examinations in appendicitis, however, are, on account of the vastness and complexity of its differential diagnostic aspect which reaches into the most difficult branches of roentgenology, decidedly beyond the ability of any clinician. Therefore, if a clinician does his own roentgen work even in these very difficult cases, the responsibility for grave diagnostic errors must rest with him. Similarly, responsibility for roentgen diagnostic mistakes in appendicitis must be shouldered by those practitioners who refer their patients to incompetent roentgenologists.

This phase of the roentgen situation where the patient's welfare is entirely disregarded by his own health custodian has gradually become so serious that there will come a time when the patient will ask and obtain full redress for such diagnostic errors for which there is absolutely no excuse.

It will probably be then that the roentgen examination in appendicitis will be automatically placed in the hands of competent roentgenologists where it exclusively belongs. And it will be then that the roentgen diagnosis of appendicitis will no more be considered "a delusion and a snare," but a diagnostic aid highly esteemed and eagerly sought by physician and surgeon for the benefit of the patient.

Conclusions

Roentgenology is the best diagnostic method in chronic appendicitis.

Its main value lies in the differential diagnosis, because the roentgen examination enables us much better than any other examination method to recognize the most important lesions which are apt to simulate the clinical picture of appendicitis.

Success in this respect calls for a systematic arrangement of the roentgenological procedure and a very careful history so that all the diagnostic possibilities will be covered. Roentgenoscopy alone is not sufficient for this purpose.

The vastness of the differential diagnostic problem makes it absolutely imperative that the roentgen examination be made by a competent roentgenologist.

If made by clinicians, technicians, or incompetent roentgen laboratories, roentgen examinations in appendicitis may lead to diagnostic errors which will prove most disastrous for the patient.

The roentgen diagnosis of appendicitis is based upon one single sign, namely, local tenderness as elicited by palpation over the visualized appendix region. All the other so-called roentgen signs are of no diagnostic value.

Evidence in regard to the appendix as furnished by the roentgenogram is of value only to the surgeon for the planning of the operative procedure.

Chronic gross anatomical changes, sometimes even of the most advanced type, occur in an appreciable percentage of clinically normal appendices.

Microscopic anatomical changes are found in practically every appendix, especially of adults. This explains the uniformly found pathology in removed appendices.

The significance of chronic anatomical changes has not yet been determined.

Anatomical changes, therefore, cannot be taken as a reliable criterion for the correctness of the diagnosis of chronic appendicitis. A much better criterion is the therapeutic result, sometimes in association with self-evident proof of inflammation as found on surgical inspection.

References

1. Assmann, H. Die Röntgendiagnostik der inneren Erkrankungen. F. C. W. Vogel, Leipzig, 1929.
2. Béclère. Présentation d'un radiogramme montrant sur le vivant l'image de l'appendice iléocaecal. *Bull. et mém. Soc. de radiol. méd. de Par.*, 1909, *1*, 192–195.
3. Bennett, W. H. The use of the x-rays in the diagnosis of appendicitis and some other abdominal conditions. *Lancet*, 1908, *1*, 1461–1465.
4. Bertwistle, A. P. X-rays in the diagnosis of chronic appendicitis. *Brit. M. J.*, 1929, *1*, 1075–1076.
5. Blake, J. A. A new method for operating upon voluminous caeca or caecum mobile. *Ann. Surg.*, 1912, *55*, 767–770.
6. Buisson, M. Diagnosi clinica e diagnosi radiologica dell'appendicite cronica. *Minerva med.*, 1931, *22*, 714–724.
7. Carman, R. D. The Roentgen Diagnosis of Diseases of the Alimentary Canal. W. B. Saunders Co., Philadelphia, 1921.
8. Case, J. T. Roentgen examination of the appendix. *N. York M. J.*, 1914, *100*, 161–167.
9. Childs, S. B. Appendicitis and lesions complicating its diagnosis. *Radiology*, 1925, *4*, 107–110.
10. Cohn, M. Der Wurmfortsatz im Röntgenbilde. *Deutsche med. Wchnschr.*, 1913, *39*, 606–608.
11. Connell, F. G. Pseudo-appendicitis. *J. Am. M. Ass.*, 1916, *67*, 335–338.
12. Czepa, A. Die Röntgendiagnostik der Appendixerkrankungen. *Wien. klin. Wchnschr.*, 1926, *39*, 1431–1432.
13. Deaver, J. B. Chronic appendicitis. *Am. J. M. Sc.*, 1929, *177*, 749–758.
14. Einhorn, M., and Scholz, T. Ueber Spasmen des Verdauungskanals und deren Diagnose. *Arch. f. Verdauungskr.*, 1926, *37*, 1–16.
15. George, A. W., and Gerber, I. The value of the Röntgen method in the study of chronic appendicitis and inflammatory conditions, both congenital and acquired, about the caecum and terminal ileum. *Surg., Gynec, & Obst.*, 1913, *17*, 418–427.
16. Gottheiner, V. Die Röntgendiagnostik der Appendix. *Ergebn. d. med. Strahlenforsch.*, 1928, *3*, 425–486.
17. Henszelmann, A. Kleine röntgenologische Vorrichtung zur Erzeugung von Wurmfortsatzbildern. *Berl. klin. Wchnschr.*, 1914, *51*, 1517.
18. Holzknecht, G. Röntgenologie des Blinddarms. *25th Kongress f. inn. Med., Wien*, 1908, *25*, 259.
19. Hüffer, B., and Kaspar, M. Praktische Bedeutung der Röntgenuntersuchung für die Diagnose der chronischen Appendizitis. *Beitr. z. klin.Chir.*, 1930, *149*, 481–500.
20. Hurst, A. F. X-ray diagnosis of gastro-intestinal conditions with especial reference to appendicitis. *Arch. Roentg. Ray*, 1914, *9*, 249–255.
21. Imboden, H. M. Roentgen diagnosis of the vermiform appendix. Am. J. Roentgenol., 1915, *2*, 581–591.
22. Jacquet, P., and Gally, L. Le diagnostic radiologique de l'appendicite chronique. *Presse méd.*, 1931, *39*, 376–379.
23. Kantor, J. L. A clinical study of some common anatomical abnormalities of the colon. II. The low cecum. Am. J. Roentgenol. & Rad. Therapy, 1925, *14*, 207–221.
24. Kantor, J. L., and Schechter, Samuel. Colon studies. V. The high cecum. Am. J. Roentgenol. & Rad. Therapy, 1928, *19*, 101–111.
25. Koerbler, G. Sur le diagnostic de l'appendicite par la douleur à la pression. *Rev. de chir.*, 1929, *67*, 347–353.
26. Kruchen, C. Chronische Appendizitis unter den Erscheinungen eines Magengeschwürs. *München. med. Wchnschr.*, 1931, *78*, 92–94.
27. Liertz, R. Die radiographische Darstellung des Wurmfortsatzes. *Deutsche med. Wchnschr.*, 1910, *36*, 1269–1270.
28. Lowsley, O. S., and Twinem, F. P. Differential diagnosis of pain in the right side of the abdomen. *J. Am. M. Ass.*, 1929, *93*, 1614–1619.
29. Moore, A. B., and Merritt, E. A. Roentgenologic diagnosis of appendicitis. *J. Am. M. Ass.*, 1931, *97*, 1456–1457.
30. Orndoff, B. H. Roentgen-ray studies of the appendix, cecum, and ascending colon. *J. Am. M. Ass.*, 1926, *87*, 1294–1297.
31. Pfahler, G. E. The roentgen rays in the diag-

nosis of appendicitis. Am. J. Roentgenol., 1919, *6*, 78–82.

32. Peirce, C. B. The undescended cecum. Am. J. Roentgenol. & Rad. Therapy, 1931, *26*, 839–860.
33. Rieder, H. Zur Röntgenuntersuchung des Wurmfortsatzes, besonders bei Appendizitis. *München. med. Wchnschr.*, 1914, *61*, 1492–1494.
34. Scholz, T. A roentgenological contribution to the diagnosis of spinal carcinoma in cases having an unrecognized primary focus. *N. York M. J.*, 1922, *116*, 566–570.
35. Scholz, T. The diagnosis of spinal metastases having a clinically latent primary focus within the chest. *Med. J. & Rec.* (*suppl.*), 1924, *20*, xx–xxiv.
36. Singer, G., and Holzknecht, G. Radiologische Anhaltspunkte zur Diagnose der chronischen Appendizitis. *München. med. Wchnschr.*, 1913, *60*, 2659–2664.
37. Skinner, E. H. The roentgenology of appendical obliteration. *J. Am. M. Ass.*, 1920, *75*, 1614–1617.
38. Spriggs, E. I., and Marxer, O. A. The examination of the vermiform appendix by x-rays. *Lancet*, 1919, *1*, 91–97.
39. Stierlin, E. Das Coecum mobile als Ursache mancher Fälle sog. chronischer Appendicitis und die Erfolge der Coecopexie. *Deutsche Ztschr. f. Chir.*, 1910, *106*, 407–476.
40. Waller, C. C., and Cole, L. G. The appendix. *Surg., Gynec. & Obst.*, 1915, *21*, 750–759.
41. White, F. W. The clinical importance of chronic changes in the appendix. Am. J. Roentgenol. & Rad. Therapy, 1925, *13*, 12–21.
42. Williams, J. T., and Slater, R. Condition of the appendix in five hundred laparotomies on patients presenting no symptoms of appendicitis. *Ann. Surg.*, 1919, *70*, 535–538.
43. Wilms. Fixation des Coecum mobile bei Fällen von sogenannter chronischer Appendicitis. *Centralbl. f. Chir.*, 1908, *35*, 1089–1091.

ROENTGEN-RAY STANDARDS AND UNITS[1]

STANDARDIZING PROCEDURE OF THE NATIONAL LABORATORIES[2]

TO FACILITATE the establishment of roentgenological standards and to assist in promoting cooperation between the various countries having such standards, the following draft memorandum on standardizing procedure has been jointly agreed upon in preliminary form by the following national institutions between which a direct comparison of roentgen-ray standards has been carried out:

National Bureau of Standards, United States of America
National Physical Laboratory, Great Britain
Physikalisch-Technische Reichsanstalt, Germany
Service d'Etalonnage de l'Hôpital St. Antoine, France.

The methods of procedure indicated are necessarily dictated by the experience of the individual laboratories, and may be subject to modification according to future advances in the science. The four laboratories will continue to collaborate in the development, maintenance and intercomparison of roentgenological standards and units, and will issue further memoranda in the future as occasion may arise. They propose to bring the present memorandum to the notice of the Fourth International Congress of Radiology at Zurich in July, 1934.

Each of the participating laboratories will sponsor publication, in its own country, of the present memorandum which will also be submitted for publication to the *Acta radiologica* in Stockholm. In the meantime, comments are invited by the participating laboratories.

I. THE MEASUREMENT OF QUALITY

(*1*) *Roentgen-Ray Quality* (*hardness, spectral distribution*).

The term roentgen-ray quality relates to the composition of a roentgen beam as determined by the spectral range and energy distribution.

In the case of *homogeneous roentgen rays*, the quality may be expressed in terms of the wave length of the radiation, measured in Angström units ($1A = 10^{-8}$ cm. = 1000 X.U.).

In the case of the *heterogeneous roentgen rays* which occur in practice, the following table outlines a convenient practical classification of quality:

Designation	Approximate Range of Applied Potential	Equivalent "End-Radiation" Limits
	kv. peak	Å
"*Ultra-soft*" (grenz) radiation	5– 20	2.5–0.6
"*Soft*" radiation for diagnostic work and superficial therapy	20–120	0.6–0.1
"*Hard*" radiation for deep therapy	120–250	0.1–0.05
"*Extra-hard*" or "*super-hard*" radiation for highest potential therapy	Over 250	Less than 0.05

(*2*) *Quality Specification.*

In medical practice it is not convenient to express the quality of heterogeneous radiation in terms of its spectral distribution. The quality may, however, be expressed with sufficient accuracy for such purposes by a statement of generating conditions and absorption data, e.g.:

(*A*) *Generating conditions.*

(a) The target material.
(b) Either the peak or r.m.s. value of the potential applied to the roentgen tube.
(c) The potential wave form (e.g., constant or pulsating).
(d) The type of filtration.

[1] Publication approved by the Director of the Bureau of Standards of the U. S. Department of Commerce.

[2] Collaborators: H. Behnken, Physikalisch-Technische Reichsanstalt; G. W. C. Kaye, National Physical Laboratory; I. Solomon, Service d'Etalonnage de l'Hôpital St. Antoine; and L. S. Taylor, National Bureau of Standards.

(*B*) *Absorption data.*

(a) Form of the total absorption curve in celluloid for ultra-soft radiation (5–29 kv.); aluminum for soft radiation (20–120 kv.); copper for hard radiation (100–250 kv.); copper or lead for ultra-hard radiation (above 250 kv.).

or (b) Frequently, it is sufficient for practical purposes to state the thickness in millimeters of the absorbing material (celluloid, aluminum copper or lead) which, by insertion in the beam of radiation, reduces the irradiation—ionometric intensity—to one-half (half-value layer).

II. MEASUREMENT OF THE ROENTGEN

Recent international comparison has shown that an agreement to within at least ±1 per cent is attainable between the standard roentgen-ray dosage measurements of the participating laboratories. The following experimental precautions should be adopted in standard measurements:

(*3*) *Roentgen-Ray Tube.*

The roentgen-ray tube should be of the hot-cathode type with tungsten target. The focal spot should be as small and as uniform as possible and the target should not be appreciably pitted. Tube current and applied potential shall be kept constant with the requisite degree of accuracy during a test.

(*4*) *Roentgen-Ray Beam.*

A diaphragm of suitable aperture shall be mounted as near as possible to the roentgen tube so that the roentgen beam utilized shall include a maximum of radiation from the focal spot, and a minimum of off-focus radiation. The uniformity of the radiation field of the test beam shall be verified by ionization or photographic methods. For measurements made at different focal distances, deviation from the inverse square law should not exceed the specified measurement accuracy. The certainty of this shall be verified by special test. Where, for practical reasons, the inverse square law does not hold, the accuracy of the results is likely to be increased by conducting the measurements as far as possible from the roentgen tube.

(*5*) *Standard Ionization Chamber.*

(*A*) *Chambers for either soft or hard radiation.*

Chambers used for measurement (in roentgens) of either soft or hard roentgen rays shall be of the open air type (free-air, Fasskammer) wherein the measured ions are produced in a column of air which is not bounded by walls. Different forms of such chambers have been found to be satisfactory by the participating laboratories. For full constructional details the several publications of the laboratories may be referred to.

The following general conditions shall be satisfied by standard chambers:

(a) The *ionization chamber* shall be of such dimensions and so constructed that:

(i) The spacing between the measuring and the high-potential electrodes is sufficient to insure full utilization of the secondary electrons;

(ii) The electric field between electrodes is sufficient to insure saturation. Chamber forms which make the attainment of saturation difficult are unsuitable;

(iii) Distortion of the electric field between the measuring and the high potential electrodes is avoided as far as possible, for example, by the use of suitable guard electrodes or a guarded field.

(b) A *chamber diaphragm* (entrant diaphragm, limiting diaphragm) shall be mounted to provide an entrance for the roentgen-ray beam to the chamber and shall conform to the following general conditions:

(i) The material out of which the diaphragm is made shall be of the minimum thickness which will permit not more than one part in

a thousand (1/1000) of the measured radiation to pass through the material itself;

(ii) The diameter of its aperture should be approximately equal to the diameter of the focal spot of the roentgen tube, but always such as to limit the beam entering the chamber to one of uniform intensity of cross section;

(iii) The aperture should preferably be cylindrical on account of the ease of construction and accuracy of measurement. A covering material such as cellophane shall not be used;

(iv) The distance of the chamber diaphragm from the measuring electrode shall not be unnecessarily large, in order that the correction for air absorption of the rays along the path of the beam between the diaphragm and the electrode shall be a minimum;

(v) Precautions shall be taken to avoid a measurable amount of scattered electrons or secondary radiation originating at the rim of the chamber diaphragm from reaching the measuring volume. For example, a second larger diaphragm whose rim is not struck by the direct beam of radiation may be placed between the chamber diaphragm and measuring electrode.

(c) The *ionization current* is preferably measured by a null method so that a distorting electric field does not exist between the guard and measuring electrodes. Radioactive compensators should be of the constant capacity type.

(*B*) *Chambers for Soft Radiation.*

For the field of diagnosis and superficial therapy (soft radiation), a chamber smaller than for the hard radiation may be used to advantage. This should, of course, agree with the larger chamber over the greatest possible quality range.

(*C*) *Chambers for Ultra-Soft and Super-Hard Radiation.*

While preliminary studies have indicated the probability that the standard ionization chamber employed for soft and hard radiation may be satisfactorily modified for use with ultra-soft and super-hard radiation, sufficient experience has not yet been gained to justify international agreement thereon.

III. CALIBRATION OF ROENTGEN DOSIMETERS

(*Roentgenometers, Dosimeters, Irradiometers, Intensimeters, etc.*)

(*6*) *Small Ionization Chambers.*

(A) Small ionization chambers such as are frequently used in practice should be so designed and constructed as to be independent of quality within the spectral range for which they are intended.

(B) Such chambers shall be equipped with suitable arrangements for checking the constancy of their calibration.

(C) There shall be no ionization in the instrument extraneous to that in the small chamber itself.

(*7*) *Calibration of Small Ionization Chambers.*

(A) Calibrations of small chambers may be carried out by either a direct replacement method, or a method depending upon the inverse square law when its validity has been proved.

(B) Approximately the same portion of the test beam should be used for both standard chamber and small chamber. This should be chosen from a portion of the main beam which is uniform.

(C) Walled chambers of the thimble type shall be completely enveloped by the test beam.

(D) Filters necessary in the calibration of small chambers shall be so placed in the roentgen-ray beam that the effect of secondary radiation therefrom is not included in the measurements.

(*8*) *Reports on the Calibration of Small Chambers.*

In reports on the above types of instruments, the participating laboratories will normally include the following:

(A) The equivalent in roentgens of the overall (or other desired) reading of the scale for each quality of test beam used.

(B) Data on the quality of each test beam used (in accordance with paragraph 7).

(C) The temperature and pressure at which the calibration was conducted, together with correction factors for other temperatures and pressures.

(D) Data on the irradiation (dosisleistung, ionometric intensity) which was used for the calibration, hence for which the calibration is valid.

(E) Data on the means provided to check the secular constancy of the sensitivity of the instrument.

Vol. XXXI, No. 6

A NEW METHOD FOR THE TREATMENT OF "BLEEDING NIPPLE" BY RADIUM IMPLANTATION*

By MAX CUTLER, M.D.†
CHICAGO, ILLINOIS

THE PURPOSE of this communicaton is to present a new method for the treatment of lesions underlying serous and serosanguineous discharges from the nipple. The method referred to consists of the implantation of removable platinum filtered radium element needles into the breast about the nipple and areola.

CLINICAL CONSIDERATIONS

The clinical problem of "bleeding nipple" may be divided conveniently into two categories as follows: (1) The treatment of breasts exhibiting a serous or serosanguineous discharge from the nipple in which there is no palpable tumor. (2) The treatment of breasts in which a serous or serohemorrhagic discharge from the nipple is accompanied by a palpable tumor.

STATEMENT OF THE PROBLEM

The syndrome which constitutes the subject of this communication, commonly referred to as "bleeding nipple," occurs in breasts which are the seat of a recognized pathological process. The pathological state underlying a serous or serohemorrhagic discharge from the nipple consists of one or more duct papillomata or of duct carcinoma. When this sign is accompanied by a palpable tumor the diagnosis is clear and the line of procedure is clarified. Under these circumstances carcinoma has become fully established and the breast must be treated for cancer according to all the criteria used in the treatment of this disease. A far more difficult diagnostic and therapeutic problem confronts us when a breast exhibits a serous or serohemorrhagic discharge from the nipple in which no palpable tumor exists. As patients are consulting their physicians earlier the majority of women who now come under observation for this sign do not harbor a palpable tumor of the breast. Thus the problem becomes more complex both in regard to diagnosis and to treatment.

It was stated above that the lesion underlying the syndrome of "bleeding nipple" is either duct papilloma or duct carcinoma. In the presence of a palpable tumor the diagnosis of carcinoma is justified unless the tumor is an unrelated lesion as, for example, fibroadenoma. The important question arises: How commonly does one encounter duct carcinoma in the presence of a hemorrhagic discharge from the nipple when a tumor cannot be palpated? In my experience this is a rare event; I have seen it in only one case. It is safe to state that the greater possibility under these circumstances is the presence of a benign lesion. This brings us to the crucial part of the problem. What is the prognosis of duct papilloma of the breast?

This subject finds itself in a state of confusion, some authors looking upon the lesion as innocent, others regarding it as of grave significance. Those who look upon duct papilloma as an innocent lesion base their position upon the experience that certain patients harboring this lesion in their breasts fail to develop carcinoma after a lapse of some years even though the condition is not treated. Those who look upon this lesion as being a grave menace point to the following evidence: (1) A certain definite proportion of breasts exhibiting a bloody discharge from the nipple ultimately become the seat of carcinoma. (2) The supervention of carcinoma upon the benign process may require many years (as long

* Read before the American Congress of Radiology, Chicago, Ill., Sept. 25–30, 1933.
† Director, Tumor Clinic, Michael Reese Hospital, Chicago; Consultant in Tumors, Edward Hines, Jr. U.S. Veterans Hospital, Hines, Illinois.

as twenty, thirty, or forty years) so that breasts in which this complication has not occurred in shorter periods are by no means immune. (It is admitted that in a certain proportion of cases the supervention of carcinoma may never occur.) (3) In the benign papillomatous state in other organs, notably the bladder and colon, the supervention of carcinoma is in the bladder—a frequent event and in the colon almost a certainty. (4) Experimental evidence, particularly with tar, proves the benign papillomatous stage to be one step in the cancer process. (5) When cancer complicates the papillomatous state in the breast the two lesions occur in the same duct unit, thus ruling out the possible fallacy that the two lesions may be accidental rather than causative.

THE PRACTICAL PROBLEM

From a practical point of view, when a patient presents herself with a spontaneous serous or serohemorrhagic discharge from the nipple and no tumor can be palpated in the breast, what is the safe and conservative procedure to adopt? It is my opinion that to permit such a lesion to go untreated is dangerous and unjustified.

The safest method to pursue when the discharge is hemorrhagic is to perform a complete excision of the whole breast without the fascia or muscles or axillary lymphatic glands. This procedure is advocated by no less authority than Sir George Lenthal Cheatle of London. In women past the age of forty this line of procedure is the safest measure to adopt. At this point the question arises as to the wisdom of attempting to perform a local excision for this condition. If a local excision is decided upon, how is the lesion to be localized and what will determine the extent of the excision?

LOCALIZATION OF THE LESION

A determination of the exact source of a serous or serohemorrhagic discharge from the nipple by clinical examination is actually a difficult matter and sometimes one impossible of execution. In the absence of a palpable lesion, careful pressure with one finger may be of some help but it is frequently unsuccessful. It is in this field that transillumination finds its greatest usefulness. I originally reported upon this method in 1929. Since this observation it has become evident to me and to others who have employed this method that transillumination is a useful procedure in the localization of duct papillomata underlying "bleeding nipple" (Ewing, Cheatle, Bloodgood, Lee and Adair).

I wish to re-emphasize that in some instances, particularly when the papillomata are very small, localization is not possible by means of transillumination. In most cases, however, careful examination of the breast by transillumination in a totally dark room reveals the presence and position of intraduct papillomata and within limits indicates the extent of their distribution.

Local excision for duct papilloma is applicable to those lesions which are situated in the region of the nipple or areola which can be localized by transillumination. This conservative procedure is especially indicated when the patient is young so that chances of carcinoma are at a minimum and because the patient prefers to avoid or refuses the removal of the whole breast.

The disadvantages of local excision for this lesion are numerous: (1) If carcinoma exists, the excision is inadequate. This objection is overcome by performing a frozen section of the removed specimen and carrying out further treatment if carcinoma is discovered. (2) Although transillumination fails to disclose but one lesion, other microscopical lesions may be present and remain outside the field of operation. (3) The papillomatous process is sometimes so widespread as to involve the entire duct system of the breast. (4) There are considerable technical difficulties in attempting to execute a local excision for duct papilloma, which may prove unsuccessful.

Thus local excision is impossible in the group of cases in which the lesion cannot

be localized by palpation or transillumination, inadequate in cases of diffuse papillomatosis, and uncertain of removing the entire lesion in apparently localized papillomata. Furthermore, it must be promptly followed by more extensive surgical measures if carcinoma is discovered in the excised specimen.

Thus we find ourselves in a rather unsatisfactory position as regards the surgical treatment of this group of cases. The wisdom of performing complete local mastectomy in older women is undisputed. But in younger women the question arises, are we justified in recommending the removal of the entire breast in the presence of a serous or serohemorrhagic discharge from the nipple for a lesion that is most likely not cancer and which, although potentially cancer, is not certain to become malignant?

THE USE OF INTERSTITIAL IRRADIATION IN CASES OF "BLEEDING NIPPLE"

I have attempted in the past to treat "bleeding nipple" by external irradiation without success. It is quite possible that the failure can be accounted for by inadequate dosage. The work of Keynes of London in the treatment of mammary cancer by interstitial irradiation has proved definitely that a certain proportion of malignant tumors of the breast can be sterilized and the patients may remain well for years. In fact, it is the opinion of Keynes that this method is as successful as surgical removal of the process. Without entering into the controversial field of the relative merits of radiation and surgery in the treatment of operable mammary cancer, it is recognized by all that Keynes has made an important contribution to the technique of irradiation of breast cancer. The important features of this method are adequate filtration, homogeneous distribution, and more particularly, the prolonged irradiation which extends over a period of seven or eight days.

I have employed the Keynes technique for inoperable and sometimes operable mammary cancer with radium element needles and also with emanation. In my opinion, the criticism of the method is the practical impossibility of sterilizing the axillary lymphatic gland area by means of interstitial irradiation with regularity and certainty. This, I believe, is the important drawback to the method. So far as the sterilization of cancer in the mammary gland itself is concerned, I agree with Keynes and regard his contribution of great value.

Considerable experience has now accumulated upon the microscopical changes that occur following interstitial irradiation of mammary cancer. Thus it is a logical step to consider that if a fully established primary mammary carcinoma can be sterilized with some degree of certainty by interstitial irradiation, a duct papilloma or multiple papillomata, or diffuse duct papillomatosis can be similarly affected by this means. This conclusion might be challenged on the basis that carcinoma cells are more sensitive to irradiation than the cells of duct papilloma, but whereas this criticism might be justified if external irradiation were employed, it can be disregarded when interstitial irradiation is the method adopted. There is ample proof available that an adequate dose of radiation administered to a papilloma by interstitial treatment results in destruction of the lesion and its replacement by fibrous connective tissue.

In view of these considerations, it seems logical to suggest the implantation of removable radium element needles for the treatment of selected patients suffering from a serous or serohemorrhagic discharge from the nipple. The method offers the advantage of avoiding the radical procedure of breast amputation for a lesion which may not be cancer and usually is not cancer when treatment is administered and which may possibly never become cancer. It is a more complete procedure than local excision as it treats the entire duct system of the breast and at the same time avoids surgical interference. It should be adequate to destroy completely an early

duct carcinoma and especially if it is so early as to fail to be accompanied by a palpable tumor.

It appears to me to offer an intermediate method between two extremes: the one, over-conservatism, consisting of no treatment, which is dangerous, and the other, complete removal of the breast, which although safe, may be considered radical for small benign lesions.

Two patients whose breasts exhibited discharges from the nipple have been treated by the implantation of removable platinum radium needles. In both instances the discharge has stopped and there is no clinical evidence of disease after twenty-four and eighteen months, respectively.

REPORT OF CASES

CASE I. The patient was a woman aged forty-five. She was married and had two children aged twenty-two and twenty-five years, respectively. She had nursed both children without difficulty. The family history is negative for cancer. She has had inverted nipples as long as she can remember. In 1927, five years previously, a hysterectomy had been performed for fibromyoma and soon afterwards she noted a clear, watery discharge from the left nipple. About two years before, roentgen treatment was administered over the breast once each week for eight weeks. This cycle of treatment was repeated over a period of two years with three to four week intervals. The exact dosage could not be determined but the skin became markedly pigmented. The discharge from the nipple was not affected. The patient was examined on January 6, 1932. Both nipples were inverted. The skin overlying the left breast was highly pigmented but intact. There were no masses to be felt in either breast. The left nipple was covered with a dry crust and a clear straw-colored fluid could be easily expressed from the nipple on gentle pressure. There was no actual erosion of the nipple, consequently Paget's disease, which had been suspected, was ruled out. Transillumination failed to show an opacity. The patient refused any form of operative intervention.

On April 25, 1932, radium implantation was performed as follows: Four platinum needles each 1.9 cm. long and containing 1 mg. of radium element, ten platinum needles each 3.2 cm. long and containing 2 mg. of radium element and two platinum needles each 4.4 cm. long, each containing 3 mg. of radium element, were distributed around the nipple and areola. The total radium content of the needles was 30 mg. and they remained in place for fifty hours. The total dose was 1,500 mg-hr. The discharge which had been present for five years promptly stopped and has not recurred up to the present (twenty-four months).

CASE II. The patient was a woman aged forty-one. She had two children. The father and one sister had died of cancer. A yellowish spontaneous discharge from the left nipple had been noted for six months. On examination, both breasts were normal to inspection and palpation. On transillumination a definite opacity was noted in the upper inner quadrant of the left areola, close to the nipple. There were no palpable masses. Gentle pressure over this area caused the escape of a clear, slightly pink fluid which on microscopical examination showed the presence of red blood cells.

On November 29, 1932, implantation of radium was executed as follows: Eleven platinum needles, 3.2 cm. in length and each containing 2 mg. of radium element and five platinum needles, 1.9 cm. long, each containing 1 mg. of radium element, were implanted in the breast around the nipple and areola for seventy-two hours. The discharge promptly stopped and has not recurred eighteen months after treatment.

CONCLUSIONS

Interstitial irradiation is suggested as a new method for the treatment of selected patients suffering from serous or serohemorrhagic discharge from the nipple. It has the advantage of avoiding the removal of the breast, especially in young women, for a lesion which although potentially cancerous is still benign.

Vol. XXXI, No. 6

ROENTGEN THERAPY OF ACTINOMYCOSIS*

By E. GERARD SMITH, B.Sc., M.D.
BOSTON, MASSACHUSETTS

MANY methods have been used in the treatment of actinomycosis. Few of them effect a cure and some of them may aggravate the infection. The results obtained by the use of roentgen rays have been considered satisfactory in some cases. In spite of the fact that roentgen irradiation was first employed in cases of actinomycosis in this country, relatively few articles dealing with this subject are to be found in the American literature. The vast majority of cases so treated have been recorded in the European journals and these are not readily available to the average practitioner. Because of these facts and because our interest in the technique of roentgen therapy was aroused by a case we are now treating, a review of the literature was undertaken.

Among the various therapeutic agents used are copper sulphate, vaccines, methylene blue, and salvarsan. All of them have lost favor, although cures have been reported as having resulted from their use. Iodides are generally used but some doubt as to their efficacy has been raised by Heeren, Jüngling, and others. These authors reported a series of cases some of which received iodides during roentgen treatment and some of which did not. The results were the same. Radical surgery including curetting is probably inadvisable.

A study of the cases reported by the authors quoted herein shows that, all other things being equal, those cases which had a minimum of surgical interference recovered more rapidly than did the remainder. Surgical procedures should probably not be resorted to except to provide drainage and to assist in definitely establishing a diagnosis. They should then be limited to a small incision, preferably a stab wound. Cases have been reported as cured by the use of radium. Unfortunately, the size of most actinomycotic lesions, combined with the scarcity and prohibitive cost of the element, makes its use impractical.

Actinomycosis was first successfully treated with roentgen rays by Harsha (1904) in this country. The case reported was that of a man aged fifty who developed a swelling at the angle of the right jaw following the extraction of two teeth. The mass was incised but did not heal and was regarded as being sarcoma. Following the administration of iodides by mouth and the use of roentgen rays the wound healed, the swelling disappeared and the patient remained well. Bevan (1905) reported several cases treated with roentgen rays. One of these had an advanced abdominal lesion. All but one of these showed considerable improvement after roentgen therapy and the administration of iodides. Iselin (1910) had observed for several years that the induration of actinomycotic lesions regressed rapidly under the influence of roentgen radiation. Levy (1913) recorded 2 cases. The first of these was cured by the use of roentgen rays alone. The second case had been subjected to many therapeutic procedures but was cured only after exposure to roentgen rays. Sardemann (1914) recorded 4 cases cured by the combined use of roentgen radiation, surgery and iodides, but credited the result chiefly to the use of roentgen rays. Since that time cases showing the effectiveness of roentgen irradiation have been published by Nordentoft (1914), Jüngling (1919) and numerous others.

ACTION OF ROENTGEN RAYS ON THE FUNGUS

Kleesattel carried out experiments in which he subjected cultures of actinomyces to roentgen rays. While he noted that ten erythema doses had no lethal effect on the organism in vitro, he also noted that a

* From the Radiological Clinic, Massachusetts General Hospital (Dr. George W. Holmes, Director).

week after irradiation the speed of growth was retarded for a period of several days. The exact action or influence of roentgen rays on the actinomyces in vivo is not known. Some authors believe that when an individual who has been receiving large doses of iodides is subjected to roentgen-ray exposures nascent iodine is set free in the tissues which in turn has a deleterious effect on the organism. Since many patients have recovered from the infection as the result of the use of roentgen rays alone, this view is rather difficult to substantiate. I believe that recovery in irradiated cases is probably due to several factors, including the beneficial effect of roentgen rays often observed in inflammations of a pyogenic nature. Another factor may be the distinct difference in the morphology of the organism in vivo and in vitro. It is quite possible that it is more radiosensitive in vivo. Many bacteriologists believe that the other organisms which are usually found in actinomycotic lesions have a synergistic action, and it may also be that roentgen rays disturb this synergy and thus permit the tissues to build up their own defence, helping in this way to effect a cure.

CERVICOFACIAL LESIONS

The cervicofacial region is by far the most common site for actinomycotic infections. Lord gives the percentage as 50 while Boyd believes it to be as high as 60. When one inquires into the history, the patient may recall having chewed grass or having used a bit of straw as a toothpick. Often the infection is coincident with, or rather follows, trauma to the region such as a slight abrasion or the extraction of a carious tooth. The lesion is similar to that of the other granulomata. A firm mass slowly develops and the surrounding tissues become "board-like" to the touch. Sometimes it is painful but not always. The skin may or may not become discolored. In many cases it takes on an angry purplish hue. After a varying length of time the mass softens, abscesses form and then break down leaving one or more sinuses. The latter usually discharge a pus which contains the typical sulphur granules. These granules are not always abundant, however, and in every suspected case a careful search should be made.

On reviewing the results of the roentgen treatment of cervicofacial actinomycosis one cannot help but regard roentgen therapy as a distinct advance over other types of treatment. This is particularly true in view of the fact that the technique used was for many years more or less haphazard. Most of the early cases were treated with superficial or long wave therapy, that is, rays generated at 95–130 kv. peak, passed through a filter of 1 to 2 mm. aluminum. A few received medium wave therapy and fewer still short wave therapy. The results in this type of lesion were fairly uniform, however, and probably because the depth of the infection is rarely more than 3–4 cm., wave length seems to make little difference. Dosage, on the other hand, is of greater importance. In a paper such as this it is impossible to chart the dosages given to all the cases which were reviewed since up to within fairly recent times many different unit doses were in use. Any common denominator must of necessity be quite arbitrary and therefore only a few of the series of cases in which dosage was given in terms of erythema doses (E.D.) will be briefly discussed.

As a general rule, those cases which received large initial doses made the most rapid and uneventful recoveries. Von Tempsky reported 36 cases, 32 of which were healed. When the patient was seen early in the course of the disease $\frac{1}{3}$ E.D. was given on three successive days. Other cases received 1 E.D. in one dose, often through several fields. The time interval between treatments varied from three to four weeks and treatments were continued as long as there was any sign of activity, often for some time after the induration and inflammation had completely disappeared. All of these cases received KI. Heeren reported 12 cases, 10 of which were healed; one died and the other improved. His

technique was similar to that used by von Tempsky and he noted that in those cases in which large intensive doses were given immediately, recovery was much more rapid than in those cases which received the same dose over a period of several days. Several of the cases which received initial doses of only 60–70 per cent E.D. did not show improvement for some time in spite of subsequent treatment. Five of his patients received KI but he was unable to note any advantage in its use. Of Jüngling's series of 13 cases one died. In this case the patient received only two doses of $\frac{2}{3}$ E.D. a month apart, followed by $\frac{1}{2}$ E.D. during the third month. All of the other cases reported by him received larger initial doses and all but one were healed. Five of his cases received KI. Eiken's experience led him to the conclusion that large intensive doses early in the course of the disease are highly desirable and the vast majority of authors concur in this view.

ABDOMINAL LESIONS

The diagnosis of abdominal actinomycosis is somewhat more difficult than that of cervicofacial lesions. Most of these patients have a history greatly resembling that of acute or chronic appendicitis and have undergone surgical operation on the basis of that diagnosis. After operation a new abscess usually forms which may likewise be characterized by "board-like" hardness and which also breaks down in the manner of cervicofacial lesions. Often the patients are operated on several times before a diagnosis is definitely established. Such were the circumstances of most of the cases recorded. As Desjardins noted,

> Many of these patients were so ill and the lesions so extensive when treatment was started that the termination was necessarily fatal.

Before drawing any conclusions from the data given in Table I it is only fair to say that most of these cases were treated with superficial therapy. Desjardins' series of cases is very interesting. Of the 20 cases in which end-results were obtained, 14 died and 4 were cured. Only 4 failed to show a definite improvement at some time. Of the 4 which were cured 3 received supplementary radium treatment, while of the 14 which died only 3 received radium. In all of these cases the entire abdomen was irradiated anteriorly through a 4 mm. Al filter and posteriorly through a 6 mm. Al filter. The rays were generated at 135 kv. peak and 5 ma. The usual dose for each field was 70–90 per cent E.D. Most of his cases received two to three series of treatments at intervals of three to four weeks. The cases reported as healed by other authors received on the average doses of $\frac{3}{4}$–1 E.D. through two or more fields at intervals of one to four weeks.

Table I

RESULTS IN CASES OF ACTINOMYCOSIS TREATED WITH ROENTGEN RAYS*

Total No. of Cases	Improved	Healed	Died
Cervicofacial Lesions			
129	12 9.36%	107 83.44%	10 7.8%
Abdominal Lesions			
45	6 13.13%	17 37.74%	22 48.84%
Thoracic Lesions			
21	2 9.52%	2 9.52%	17 80.92%

* These cases were gathered from the literature. Approximately 60 per cent of those listed as healed were followed for a period of one or more years after treatment.

THORACIC LESIONS

The mortality of those patients with thoracic lesions is the highest of the three types considered. As was the case in abdominal lesions the majority of patients were treated with superficial therapy which may account to some extent for the poor results. Most of them had reached chronicity when roentgen treatment was started. It is interesting to note that the one case which was healed in the series reported by Desjardins received a larger dose of roentgen rays than did any of the others. Both of the patients treated in this

clinic improved. One of them died at the age of seventy-five in the course of the disease, two years after the inception of roentgen treatment. It is quite possible that heavy doses of deep therapy will improve the prognosis of thoracic lesions.

REPORT OF NINE CASES

Since 1919, eighteen cases diagnosed as actinomycosis have been treated with roentgen rays in this clinic. The diagnosis of actinomycosis is not an easy one and often several laboratory examinations are necessary before the organism is found. The diagnosis of the cases reported was confirmed by one or more microscopic examinations.

CASE I. (No. 11570.) Male, aged sixty-six.

Three weeks before admission to the out-patient department on October 13, 1931, the patient had had two lower left molars extracted. Immediately thereafter his left lower jaw began to swell and became tender, hard and painful. On incision, typical actinomyces were found. There is no record of the use of KI. He received 800 r(200 kv.p., filter 0.5 mm. Cu. plus 4 mm. celluloid) in daily doses of 200 r. When seen again in a month later he had improved markedly and the wound had closed. He was then given 400 r and 200 r on the following day. On April 27, 1932, he was in excellent condition and when last heard from on June 6, 1933, there had been no recurrence.

Comment. The treatment given in this case was adequate and the result satisfactory.

CASE II. (No. 9681.) Male, aged twenty-six.

The patient was admitted to the out-patient department on October 3, 1928. Four weeks previously the patient had had a tooth extracted followed by swelling of the right lower jaw three weeks later. He was advised to use hot poultices but the swelling enlarged and became very hard and painful. It was incised on November 21, 1928, but did not improve. On re-incision on December 22, 1928, actinomyces were found in the pus and KI was given. On January 2, 1929, the patient was referred to the roentgen therapy department and received 200 r (200 kv.p., filter 0.5 mm. Cu plus 4 mm. celluloid). This dose was given at weekly intervals for one month. When seen again on March 7, 1929, there was a marked improvement and the treatment was repeated. On September 18, 1929, no palpable masses were to be found and on January 29, 1930, he was discharged as cured.

Comment. The amount of radiation given this patient appears to have been adequate. Had treatment been started earlier the period of convalescence might have been somewhat shorter.

CASE III. (No. 5717.) Male, aged fifty.

The patient, a farmer by occupation, was admitted to the out-patient department on October 9, 1922. Three weeks before admission, his right jaw had become swollen, tender and painful. A week after onset he had all his teeth extracted but the mass continued to enlarge and was incised several times by his physician without relief. When seen at this clinic the patient had two large fluctuant areas over the right jaw and two scars in the same region. On aspiration of pus typical actinomyces were found. He was given KI and on October 27, 1922, received 1 E.D. (about 300 r, 100 kv.p., filter 1 mm. Al). When last seen on November 10, 1922, the mass was greatly reduced in size and the patient's condition had improved.

Comment. The treatment in this case was inadequate and should have been started earlier. Follow-up is incomplete.

CASE IV. (No. 11641.) Male, aged twenty-seven.

In February, 1931, the patient noticed a swelling of his right jaw and had a crown removed from a tooth. The swelling grew larger and during the following month was incised three times without relief. When seen at this clinic on December 3, 1931, KI was given and the patient received 300 r(200 kv.p., filter 0.5 mm. Cu plus 4 mm. celluloid). This was repeated on the following day. When seen again on January 21, 1932, the lesion had improved considerably and the above treatment was repeated. When last heard from on May 13, 1932, the lesion had healed without any sign of recurrence.

Comment. The follow-up in this case is incomplete. The progress of the case while under observation was satisfactory.

Case v. (No. 7597.) Male, aged twenty-six.

Six months before admission on November 21, 1925, the patient noticed a slight swelling on his right jaw. This rapidly grew larger and became hard and painful. It was incised by his physician but did not subside. When seen at this clinic actinomyces were found in the pus discharging from a sinus. KI was prescribed and the patient was given about 100 r (130 kv.p., filter 3 mm. Al plus 4 mm. celluloid) at weekly intervals for three weeks. A month later the induration had practically disappeared and on February 20, 1926, he was given 150 r. Improvement continued. On May 1, 1926, and on May 18, he received about 60 r but still showed some evidence of inflammation. When last seen on January 21, 1927, the patient had a small persistent sinus. Improved.

Comment. The dose given in this case was also inadequate, in the light of our present conceptions. The follow-up is incomplete.

Case vi. (No. 12676.) Female, aged nineteen.

Early in January, 1933, the patient noticed a swelling of the right side of the lower jaw which prevented her opening her mouth. Hot fomentations were applied without relief. About the middle of February the two lower right molars were extracted and a small fistulous opening was found inside the mouth near the angle of the jaw. A few days later the outside of the swelling was incised and a considerable amount of pus discharged. A second incision was made two weeks later. The lesion was suspected of being actinomycotic and on March 22 she was given 370 r (200 kv.p., filter 0.5 mm. Al plus 4 mm. celluloid) and 250 r on the following day. A reaction ensued and a few days later three small abscesses were opened. Microscopic examination showed typical actinomyces. On April 18, 1933, a roentgenogram showed involvement of the inner border of the ramus of the mandible. A week later an incision was made along the zygomatic arch and a considerable amount of pus drained away. A week later an abscess on the forehead was opened with the same result. On May 11, the patient was given 350 r and again on the following day. When seen again on June 8, 1933, the wounds had almost healed and there was practically no discharge. The treatment was repeated. At present this patient is doing very well and all signs of induration and inflammation are gone.

Comment. This case demonstrates the possibility of a severe reaction after the first roentgen treatment. It also shows the importance of using fields of ample size. The field for the first treatment was 10 ×10 cm. All subsequent treatments were given through a 15×15 cm. field. The result so far is satisfactory.

Case vii. (No. 5749.) Male, aged twenty-eight.

Five weeks prior to admission to the outpatient department on November 1, 1922, the patient noticed a mass at the angle of the left jaw. The mass rapidly increased in size and an abscess formed which broke down leaving a fistula. KI was prescribed and he was given about 300 r (100 kv.p., filter 1 mm. Al) at intervals of a month for three months. A month after the last treatment the lesion had healed and there was only a slight infiltration. He was discharged on April 13, 1923, as cured. When last heard from on June 7, 1933, there had been no recurrence.

Comment. The result in this case was satisfactory.

Case viii. (No. 9370.) Male, aged seventy-three.

The patient was admitted to the hospital on July 5, 1928. Nine months before he noticed a mass just to the right of the lower end of the sternum. The mass enlarged and broke down leaving multiple sinuses. Roentgenograms showed a homogeneous density of the right lower lobe and limitation of motion of the right diaphragm. He had a productive cough and blood-streaked sputum. On July 14, 1928 and on the following day he was given about 275 r (200 kv.p., filter 0.5 mm. Cu plus 4 mm. celluloid) anteriorly and posteriorly through a 20×20 cm. field. When seen a month later he had improved somewhat and on this occasion actinomyces were found in the pus. He was then given about 550 r anteriorly, laterally and posteriorly. When seen again at the end of another month improvement had continued and the last treatment was repeated. Two months later he had gained weight and his cough had stopped. He was then given about 275 r anteri-

orly on each of three successive days. Two months after the last treatment the sinuses had stopped draining, and he was given two doses of about 275 r. The patient did not return. A letter from a relative stated that the patient died "in the course of the disease" in May, 1930.

Comment. The actual cause of the patient's death is not known to us. While under our observation his progress was fair but in the light of our present knowledge the doses and the size of the irradiated areas should have been larger.

CASE IX. (No. 6489.) Male, aged thirty-one. In April, 1923, the patient was operated on for appendicitis. The wound continued to drain and an abscess formed which was incised during July of the same year. A short time later a second abscess formed which was also incised and was found to involve the entire right iliac fossa. In October a third abscess extending to the liver was opened and actinomyces were found. On February 27, 1924, the patient was given about 350 r (200 kv.p., filter 0.5 mm. Cu plus 4 mm. celluloid), through a 20×20 cm. field and 175 r on the following day. He grew rapidly worse and died shortly thereafter.

Comment. The poor condition of the patient when treatment was started precluded the use of large doses of roentgen rays. The effect of large doses in a case as advanced as this is highly problematical.

CONCLUSIONS

1. Roentgen treatment is a distinct advance in the therapy of actinomycosis.

2. Iodides may be used in conjunction with roentgen therapy. While most authors do not believe that iodides hasten recovery when roentgen therapy is used, they may still be of some help since cures have been attributed to their use.

3. Most authors believe that surgery should not be employed except to provide The fields should be quite large since most actinomycotic lesions are of the burrowing type. A superimposed reaction of an inflammatory type may result, but as a rule this subsides in a short time.
drainage, if this is not already established, and to aid in diagnosis.

4. It is possible that deep roentgen therapy will better the prognosis in cases of deep-seated abdominal and thoracic lesions.

5. The dose given should be as large as possible without danger of permanent damage to the skin. The latter may be disregarded to some extent in older people and larger doses given. The optimum dose is 800 r of deep therapy (1 E.D.) generated at 200 kv. peak, using a filter of 0.5 mm. Cu plus 4 mm. of celluloid, or the equivalent in some other filter. This dose should be given at intervals of one to two weeks through as many fields as are necessary.

REFERENCES

1. BECK, A. Die Bedeutung und die Probleme der Strahlentherapie in der Chirurgie, etc. *Strahlentherapie*, 1925, *19*, 232.
2. BEVAN, A. D. Actinomycosis. *Ann. Surg.*, 1905, *41*, 641–654.
3. BOYD, WILLIAM. Surgical Pathology. W. B. Saunders, Philadelphia, 1929, pp. 128–132.
4. BROCKMAN, R. ST. L. Actinomycosis of the right iliac fossa. *Brit. J. Surg.*, 1922–1923, *10*, 456–465.
5. BRUNZEL, H. F. Kasuistischer Beitrag zur Behandlung der Aktinomykose mit Röntgenstrahlen. *Strahlentherapie*, 1925, *6*, 253–256.
6. BRUCE, H. A. Actinomycosis. *Ann. Surg.*, 1923, *78*, 294–299.
7. DESJARDINS, A. U. Radiotherapy in actinomycosis. *Radiology*, 1928, *11*, 321–332.
8. DESJARDINS, A. U. Personal communication.
9. DITTRICH, R. Die Röntgenbestrahlung der Gesichts- und Halsaktinomykose. *Med. Klin.*, 1920, *16*, 394.
10. EIKEN, TH. Ueber die Röntgenbehandlung der Aktinomykose. *Acta radiol.*, 1926, *6*, 391–398.
11. GRÜNTHAL, J. Zur Behandlung der Bauchaktinomykose mit Röntgenstrahlen. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1927, *36*, 1085–1090.
12. HARSHA, W. M. Actinomycosis of the jaw. *Ann. Surg.*, 1904, *39*, 459–460.
13. HEEREN, J. Zur Bestrahlungstechnik der Aktinomykose. *Röntgenpraxis*, 1929, *1*, 475–491.
14. ISELIN, H. Von der Behandlung der Knochen- und Gelenkstuberkulose mit Röntgenlicht. *Deutsche Ztschr. f. Chir.*, 1910, *103*, 483–515.
15. JENSEN, V. W., and SCHERY, C. W. Actinomycosis treated with methylene blue and roentgen ray. *J. Am. M. Ass.*, 1920, *75*, 1470–1473.

21. Koester, E. Ueber Aktinomykose der Blasenwand. *Deutsche Ztschr. f. Chir.*, 1923, *181*, 60–65.
22. Levy, R. Röntgenbestrahlung der Aktinomykose. *Zentralbl. f. Chir.*, 1913, *40*, 121–122.
23. Lichterfeld. Ein Beitrag zur Primären Lung-
16. Jüngling, O. Zur Röntgenbehandlung der Aktinomykose. *Beitr. z. klin. Chir.*, 1919, *118*, 105–125.
17. Jüngling, O. Röntgenbehandlung der Aktinomykose der Kopf- und Halsgegend; die Methode der Wahl. *München. med. Wchnschr.*, 1919, *66*, 720–721.
18. Kaplan, I. L. Ein seltener Fall von Aktinomykose-Appendicitis. *Arch. f. klin. Chir.*, 1924, *128*, 410–416.
19. Kazda, F. Einiges über Aktinomykose, ihre Klinik und Therapie, neue therapeutische Bestrebungen. *Deutsche Ztschr. f. Chir.*, 1920, *156*, 312–377.
20. Kleesattel, H. Zur Frage der Röntgenempfindlichkeit des Strahlenpilzes. *Strahlentherapie*, 1924, *17*, 390–394.
enaktinomykose. *Deutsche med. Wchnschr.*, 1922, *48*, 801.
24. Lord, F. T. The etiology, pathogenesis, and diagnosis of actinomycosis. *Med. Clin. N. America*, 1933, *16*, 829–844.
25. Mattson, W. W. Human actinomycosis, with special reference to source and mode of infection. *Surg. Gynec. & Obst.*, 1922, *34*, 482–494.
26. Melchior, E. Klinische Erfahrungen über kombinierte Jod-Röntgentherapie der cervicofacialen Aktinomykose. *Berl. klin. Wchnschr.*, 1916, *53*, 586–588.
27. Nordentoft, J. Several cases of actinomycosis, with reference to experiment in x-ray treatment. *Nord. Tidsskr. f. Terapi*, 1913–1914, *12*, 272–276.
28. Prikul, A. Ueber die Behandlung der Aktinomykose mit Röntgenstrahlen. *Deutsche Ztschr. f. Chir.*, 1921, *166*, 414–421.
29. Sardemann, E. Ueber die Behandlung der Aktinomykose mit Röntgenstrahlen. *Beitr. z. klin. Chir.*, 1914, *90*, 157–167.
30. Sattler, E. Ueber die Bauchaktinomykose. *Wien. klin. Wchnschr.*, 1923, *36*, 714–715.
31. Steinkamm, J. Die Strahlenbehandlung der Aktinomykose. *Strahlentherapie*, 1921, *12*, 512–516.
32. Steinke. Actinomycosis cured by the roentgen ray. *J. Cutan. Dis. incl. Syph.*, 1918, *36*, 187–188.
33. Stocker, H. Die Behandlung der Aktinomykose und ihre Resultate. *Deutsche Ztschr. f. Chir.*, 1931, *230*, 169–181.
34. von Tempsky, A. Resultate der Röntgentherapie bei der Strahlenpilzerkrankung. *Beitr. z. klin. Chir.*, 1927, *139*, 207–216.
35. Wakely, C. P. G. The treatment of actinomycosis by x-rays with a report of nine cases. *Arch. Radiol. & Electroth.*, 1923, *28*, 129–135.
36. Wickhoff, M. Demonstration eines Falles von Röntgentherapie bei Aktinomykose. Heilung. *Wien. klin. Wchnschr.*, 1917, *30*, 26.
37. Zeisler, J. Notes on a case of actinomycosis. *J. Cutan. Dis. incl. Syph.*, 1906, *22*, 510–512.
38. Zwerg, H. G., and Wissmann, W. Ergebnisse der Röntgenbestrahlung pyogener und aktinomykotischer Prozesse. *München. med. Wchnschr.*, 1931, *78*, 1345–1346.

RADIUM SALTS AND EMANATION*

By FREDERICK B. FLINN
DeLamar Institute of Public Health, Columbia University
NEW YORK CITY

THE destructive action of radioactive salts on tissue, whether ingested per os, inhaled in the form of dust, injected intravenously or subcutaneously, is now well established. The action is physical rather than chemical, and the disintegration rate being unaffected by temperature or pressure and knowing where the salts are deposited, one can predict with some assurance where to look for damaged tissue. The salts, because of their relationship to the alkaline earths, regardless of how ingested, are deposited mainly in the skeleton. Smaller amounts—traces—are found in other tissues, especially the liver, kidneys and lungs. The greatest destructive action is on the bone cells which, as the case progresses, are killed, and if infection were avoided and the bone marrow not acted on because of its proximity to the bone, the entire skeleton would become a mass of dead tissue, which would gradually be absorbed. This is indicated in the roentgenograms taken at different periods during the progress of the disease. A biopsy specimen removed in the case of nonunion after four years of a spontaneous fracture of the femur showed on gross examination that one surface was covered with fibrous tissue. Opposite this was some of the yellow marrow, and slanting across from one surface almost to the other was a slit-like separation. The microscopic examination showed that the slit-like area was lined with a dense and relatively avascular fibrous tissue. This rested upon very dense bone which showed little tendency to form new bone where the fibrous tissue joined it. There was little or no evidence of bone necrosis. The tissue in the marrow spaces seemed to be well vascularized, and aside from an increase in fibroblasts showed little change. Other sections taken at autopsy of rarefaction in the skull showed the area to be filled by a rather compact replacement connective tissue, containing many delicate fibers, but very few nuclei. About the periphery of the area, within the connective tissue a few bone spicules were seen. The margin of the intact bone where it bordered on the area of replacement connective tissue appeared irregular and ragged. In spite of the irregularity the line of demarcation was sharp between normal appearing bone and replacement connective tissue. There was no evidence of an exudative or cellular inflammatory reaction. The impression was that an agent locally situated was capable of slowly melting away the bone tissue that surrounded it, with gradual enlargement of a roughly circular area of bone destruction with replacement by fibrous tissue. Evidence of fractured ribs, union taking place in some, is noted in some cases. At necropsy both in humans and in animals the bone is very brittle, and in the case of animals it is often difficult to remove the ribs without fracturing them. Roentgenograms, as a rule, do not reveal the true bone condition until the absorption process has progressed somewhat.

There are apparently two types of stimulation present. For a very short period one may find evidence of a stimulation such as is suggested by the polycythemia present, followed by a destructive action. We have seen this same type of stimulation in our tissue culture work. This stimulation might be explained by the over response of the organism to a destructive action. The other type of stimulation appears where the quantity of radioactive material is relatively small and again stops when a lower limit is reached. Whenever I have seen evidence of this second type of stimu-

* Part of the expenses of this work was paid by the Josiah Macy Foundation.

lation in the roentgenograms, the attending physician has been warned that there is a possibility that a tumor might develop and in each case so far the prognosis has been correct. Whether the effects seen in the roentgenograms are those of true stimulation is perhaps as yet undetermined. We expect shortly to publish a preliminary report on this stimulating effect as seen in our tissue culture work.*

The main destructive action is due in all probability to the alpha particle as its ionization and photographic action is 10,000 to 1,000 to 1 as compared to the action of the beta particle and gamma ray respectively. Because the penetrating action of the alpha particle is very slight the actual number of ions formed by the other two may be almost as great by the time they are stopped. We are referring to the number formed per cubic millimeter. There are four products emitting alpha particles in the disintegration series from radium to radium C inclusive; 14.8×10^{10} alpha particles are given off by 1 gram of radium and the product radon, radium A, B and C commonly considered as being in equilibrium with it. According to Swann and del Rosario's[1] work the chance contact of a single alpha particle with the nucleus of the cell is sufficient to cause death.

Thorotrast has been advocated by persons not familiar with the positive action of radioactive material to bring out more clearly the spleen and liver in roentgenograms. This compound is retained almost entirely by the liver and spleen and according to experimental data remains in the body more or less indefinitely. Thorotrast is an emulsion of thorium dioxide and can be shown to be radioactive by means of the gamma-ray instrument. The state of activity is not altered by being injected into the body which is easily demonstrated by electroscopic tests on animals which have been injected with this material several months previously. Because it is mainly retained in the liver and spleen, one would not expect to find bone changes taking place as in the case of soluble radium salts but in the organs of retention and blood, and it is here that it is found. It must be remembered in looking for damaged tissue that it is not a matter of days but of months and years, depending on the quantity present. The radioactivity in this case is small, around 0.2 of a microgram, but one is dealing with a radioactive substance that gives off five alpha particles to the four of radium and these particles on the whole have a greater penetrating power than the alpha particle of radium. The radioactive material is retained in about 1.5 kilograms of tissue which if distributed in the same ratio over the entire body of a 70 kilogram man would be equivalent to 9.4 micrograms of radioactivity. Also it should be recalled that we do not find any such concentration in the liver and spleen in our industrial cases as would be present in one of these organs of retention. We have found that 9.4 micrograms spread over the entire skeleton will produce ill effects after ten or fifteen years.

The question is constantly being asked, what is the effect of the small amount of emanations which one may breathe in a radium laboratory or ingested through the drinking of radioactive waters either naturally or artificially activated? The danger from emanations in the radium laboratory is recognized and every effort made to overcome the hazard by proper ventilation. So far we are lacking any scientific evidence or any other evidence of deleterious effects from drinking these waters. There is evidence of red-cell fragmentation when a large amount of emanation is taken into the body. This effect is also observed when radium salts have been injected intravenously in comparatively large amounts. Colwell and Russ[2] have shown that the red cells of human blood are hemolyzed much more easily by the alpha rays than by the gamma and beta rays. No hemolysis was observed by them with the last two rays with quantities "very much larger than the ionizating effects of these rays

* Recent work indicates that there is no direct stimulation of tissue exposed to radium salts.

would have indicated as being sufficiently large to produce hemolysis." This observation indicates that the red cell for some reason or other has an affinity for the alpha particle, an observation also suggested by the fact that the coefficient of absorption of these particles by the blood depends on the number of red cells present. If it were not that we are dealing with an inert gas which does not enter into any chemical combination we would think that the same reaction took place with the phosphate in the membrane of the cell as has been shown to be the case with lead. However, in the experiments of these investigators their citrated blood was exposed to 0.52 millicurie per cubic centimeter and showed no hemolytic effect for six hours and this amount is far greater than would ever be present in the drinking waters.

The development of lung carcinoma among the miners of Joachimstal and Schneeberg is suggestive of the action of the alpha particle on tissue. Unfortunately it has been impossible to get absolute proof that it is due entirely to the emanations which vary from 4 to 15 mache units per liter of air, and it has been as high as 52 in some areas. The dust of the mines contains cobalt, nickel, arsenic, lead, and uranium, and some of these have been spoken of as being the etiological agents of cancer. We will discuss this phase of the problem later in this paper.

One reason why ill effects from continually ingesting activated waters may have remained undetected is due to the fact that we have had our minds occupied by the bone changes which have been associated with radium poisoning cases among industrial workers. It is much the same situation as existed when we were looking for evidence of overexposure in the blood of these workers such as has been found to occur from an overexposure in the external use of roentgen and radium therapy. These blood changes do not appear in industrial workers until the disease has progressed toward the terminal end. The average physician knows very little or nothing whatever about radium poisoning, and the effects noted might be assigned to other causes. As has been stated, if we know where radioactive deposits are located, we can predict with considerable certainty what organs will show lesions. It is my opinion, based on my own observations, that if we find any ill effect from ingestion of activated waters it will be discovered in the kidneys or bladder—possibly in the lungs.

It is necessary to review rapidly some of the facts concerning radioactive materials in order that one may understand this problem of emanations and its action on tissue. According to Rutherford and others[3] a radioactive substance is constantly undergoing a spontaneous disintegration giving rise to the formation of a new atom distinct in physical and chemical properties from its parent substance. Radium only emits alpha particles to form a new atom called radon, an inert gas obeying all gas laws. The alpha particles, however, are retained to a large extent by the parent radium in equilibrium, the number of escaping particles depending on the form in which radium is present. In the case of radium sulphate only 5 per cent escapes while in the carbonate salts approximately 75 per cent is liberated. To get the maximum amount of emanation radium chloride in solution is used. These alpha particles in the form of a gas called radon or emanation reach equilibrium with the parent radium and in turn arrive at an equilibrium with its own disintegration products in about forty days. The disintegration products of emanation A, B and C are spoken of as active deposits of quick change inasmuch as they come into existence and disappear in a little over four hours if no emanation is present. Radium B and C emit beta and gamma rays and radium C alpha particles in addition. Because of the retention of emanation by the parent substance these disintegration products give rise to the beta and gamma rays which we so commonly associate with radium. If radium is heated

high enough it loses its activity because of the expulsion of the radon and does not reach its full radiation power until it is again in equilibrium with radon and its disintegration products.

Radon is the only *gaseous* product in the disintegration series ending in inactive lead. From radium A down we are dealing with solid substances so that any action is local and effects are not produced over the entire system as is the case with radium itself. Radon expelled by the radium deposit is carried all over the body in the blood stream, is absorbed by body fluids and is eliminated mainly through the lungs. However, new emanations are constantly being formed and the body is kept more or less in equilibrium with its radium deposits. In other words, the entire body is constantly being bathed with radioactive material.

The solubility of emanation in blood serum is a coefficient of 0.168 at 37° C., which is slightly higher than distilled water. And for red corpuscles 0.298 in a physiological saline solution. The coefficient of circulating blood is somewhere between 0.29 and 0.32, depending on the number of red cells present. The presence of lipoids influences its solution in body fluids. One of the most interesting properties of radon is that it creates an induced radioactivity by attaching itself to bodies present. This induced radioactivity is temporary and is tied up with the products of decomposition of radon on the exposed surface. One explanation of why we have had so many cases of jaw necrosis among our industrial cases in addition to that of bacterial invasion and their action on devitalized bone is that one means of elimination is through the salivary glands and also, as in the case of lead, we may get some concentration around the gums. The great activity of the saliva escaped me until recently when I was conducting some tests.

To determine what effects radioactive waters may have on the body one must first find out how long the tissues were exposed to the emanations. For this purpose I made use of an apparatus which was so designed that there was no possibility of any soluble radium salts coming in contact with the water. The instrument is capable of delivering 10,000 mache units per liter per day. For my observations I drank 200 c.c. of activated water containing 13,000 mache units per liter. The average results of the tests on my expired air taken half an hour after drinking the water showed that 90 per cent of the emanations had disappeared and over 98 per cent in an hour and a half after drinking the water. At the end of five hours one could not obtain any indication of any radioactivity by means of the alpha-ray instrument. The rate of elimination will vary according to the activity of the subject and the depth of breathing. One can safely say from my own, as well as the observations of others, that the emanations will disappear from the expired air within three or four hours after the water has been drunk. These observations indicate that the chance of any large amounts of active deposits being formed in the body is very slight. Again radium B and C, being isotropes of bismuth and lead respectively, behave chemically as these metals do and a large percentage of these deposits formed in the tissues would be eliminated in the same manner as they are. What appears to me to be the hazard from this type of exposure lies in the statement that the urine still remains active the next morning. Sonntag[4] reports that a colleague of his who took the cure at Spa Bramback was able to detect radioactivity in the urine fourteen days after his return home. I cannot bring my observations into agreement with this statement as my own findings on the urine of persons who had taken 10,000 mache units daily for over a year, but who had stopped drinking the water four days before the examination, were negative. The urines of 18 persons were examined. However, we have retained these specimens of urine which have since been evaporated to dryness with the purpose of again examining them after a year to see if there is any evidence of the presence of the long life

deposit radium D with its disintegration products.* The urine of our industrial radium poisoning cases shows considerable radioactivity in the alpha-ray instrument after evaporation but this disappears in a few hours, and is due to the active deposits of quick change A, B and C. After this time the activity is extremely small and is very difficult to determine and is due to the excretion of radium salts.

It is generally agreed that about 1 per cent of the emanations in the ingested waters decays in the body and is not eliminated by means of the lungs. This means that the active deposits of quick change will have been succeeded by the deposit of long life radium D whose half period is estimated at twenty-five years. According to tables on radioactivity by Meyers and Schweidler, radium D will come into equilibrium with radium E in fifty days and with radium F in 800 days. By a daily intake of 10,000 mache units per day the body might contain at the end of a year, not allowing for excretion of these salts, 8.14×10^{-12} grams radium D; 6.72×10^{-15} grams radium E; 0.74×10^{-13} grams radium F. These figures are entirely theoretical and no one is yet in position to say how much of the long life product will remain in the body after drinking a known strength of activated water over a definite period of time. While studying the problem of activated waters I examined 18 persons who had been drinking 10,000 mache units daily for over a year. They were instructed not to drink any of the water for four days previous to examination. The usual tests with the gamma-ray instrument and on the expired air were made and in addition a sample of urine was examined. The results were all negative but this is not surprising when we consider the type of deposits which would be present in the body. Radium D, radioactive lead only gives off a very soft beta ray with very slight penetrative power, and E a beta ray, F (polonium) an alpha particle. Radium D and E also give off a gamma ray which would be hard to detect because the quantity present would be so small. This makes it evident that we must use some other methods for detecting radioactivity of the body in making a study of this problem. Work by Lattès and Lacassagne[5] show that radium E and F are eliminated slowly through the kidney so that the quantities given are maximum.

It has been found that when radium E is injected into an animal the kidneys and spleen retain the largest quantity of active material and after these organs, the liver. This results in blood changes and nephritis. The largest amount of radium F is found in the villosities of the placenta, cortex of the kidney and spleen. There is also an appreciable quantity in the liver and lungs, with only traces in the bones. There is no explanation offered as to why these compounds are located in the interior of each of these organs except that they are the organs which are concerned with the removal of foreign substances from the blood. Chronic nephritis results from the ingestion of small doses of polonium. Because of the results above noted one would feel that the continual drinking of activated waters might produce ill effects. Because the amounts are very small the patient may live many years, and nephritis being one of the diseases commonly reported in persons of middle age, the cause might easily escape the attention of the attending doctor. It has been my observation that persons resorting to this remedy are usually middle aged. Certainly persons suffering from kidney or bladder trouble should not be exposed to this treatment. Just what the hazard is in drinking these waters is difficult to tell and will require careful case histories and autopsies with the above knowledge in mind. At this time no one is qualified to say.

The apparatus used for activating waters in this country are put on the market by persons who are, as a rule, ignorant of the subject of radioactivity and who quote phrases from the literature to hide this

* Tests now made show presence of polonium in these samples.

ignorance. Many times they have taken sentences from articles on this subject and used them in such a way as to give an impression which the author had never intended. The instructions are usually given to drink the water freely, not only by the person who is supposed to need it, but each and every member of the family. The number of diseases it is supposed to help, to say nothing of rejuvenating, is appalling. Their literature shows also a woeful ignorance of the manner of treatment which is carried out in Europe. There I found that the clinics and hospitals using this method of treatment give the entire quantity of emanation in about 4 ounces of water a day. The treatment is kept up from six to eight weeks and then stopped for four to six months, a procedure which is insisted upon as being necessary to get the best results; whether the treatment is renewed or not depends upon the symptoms. They do not claim to cure the whole list of complaints, but do find that it is most useful in rheumatic conditions, gout and certain nervous complaints. This method removes the argument that the good results claimed for this treatment are due to the quantity of water drunk.

The feeling is universal in the countries which I visited that activated waters should only be dispensed under the guidance of a physician. "The German Balneology Society deemed it its duty, especially in view of the exceedingly pronounced activity of quacks in the domain of radium therapy to draw attention to what physicians who are scientifically and clinically engaged in radium treatments have repeatedly pointed out: that Radium is a strongly effective remedy which should be prescribed only by a physician, and when taking these cures in places away from radioactive spas, the water should be dispensed only by apothecaries. In this way injuries would be positively avoided."[6] I believe it would be well to add to this statement that only physicians who have made a study of radium and have been especially trained in that science should use it. It is also my firm feeling that a bill should be passed in Congress, if possible, forbidding the sale of any radium apparatus or preparation except on the prescription of a qualified physician. The chance of any ill effects would be very much reduced if the treatments were carried on as in Europe. It is one thing to take a cure for from six to eight weeks, and another to continually drink such waters over a period of years. I am not concerned with the therapeutic value of these treatments.

Attempts have been made to answer the problem of activated waters by referring to the cases of lung carcinomas occurring among the miners of uranium ores. The problem of these miners is somewhat different in its aspects and is further complicated by the presence of mineral dusts already mentioned as well as the humidity and temperature resulting in respiratory conditions. These miners work seven hours a day in an atmosphere containing on the average about 10 mache units per liter of air. A man working in this type of operation will breathe approximately then 26 to 43 liters of air per minute. Experiments indicate that the blood will arrive at equilibrium in about half an hour and is reduced to 10 per cent at the end of an hour after being removed from the exposure. Beutel and Woldrich[7] report that in their case of a miner from Joachimstal the expired air contained 0.04 mache units per liter and the blood below 5 mache units. Unfortunately no electroscopic tests were made of the whole body, nor are we informed of the radioactive contents of various organs at autopsy if one was made. These data are absent in all cases reported so far except in the lung tissue. The report on the lung tissue in the other cases shows no activity and the absence of all minerals present in the dust of the mines. If the man were absent from work for some time this would not be surprising as all of these minerals are soluble in the body fluids and would soon disappear from the lungs. Not having all the facts at hand it is hard to say, but one would suspect that radioactive

dust had been deposited in the body of the case reported by Beutel and Woldrich.

There are about 5 liters of blood in the human body and the lungs will contain 3.5 liters of air under normal conditions. This means that when the body comes to equilibrium with the atmosphere of the mines the lungs will contain 35 mache units, the blood 50 and, allowing for the water content, the rest of the body 220 mache units; 5.12 per cent of the radon decays in seven hours. It must also be remembered that the 35 mache units in the lungs are disintegrating at the same rate and the products of decay are at least temporarily being retained in the lung tissue. While one exposure may not amount to anything the daily exposure for 300 days a year over a period of thirteen to twenty-three years might easily produce irritating effects that may result in carcinoma.

The previous work by Lacassagne and his coworkers[8] has shown that even when radium E and F are injected part of the elimination takes place by means of the lungs, and in their work the lungs were found to be radioactive at the death of the animals. Another interesting point brought out by their work was the fact that when a combination of radium E and bismuth was injected the rate of elimination is slower than when radium E is injected alone. Italian workers[9] have shown that cobalt activated by emanations is more toxic to animals than cobalt alone. The presence of the emanations appears to assist in the absorption of metals by hastening their solution; that when animals breathing emanations are injected intravenously with cobalt, cobalt is fixed in small quantities in the lung protein. Cobalt and bismuth, as well as other metals, are present in the mine dust.

We have long known that alpha particles are irritating to the skin and if exposure is kept up for a long enough time may produce a keratosis. In all my work I have observed that it is a matter of quantity times time to produce any effect. If the quantity is small it may be a matter of years and not months before the pathologic condition arises. It may be with emanations as with carbon monoxide, the body is never entirely free from it when exposed daily to small amounts. To determine what effect emanations might have on lung tissue we confined 14 guinea pigs in a glass enclosure provided with ventilation, continuously for nine months. The air of the enclosure contained 3,500 mache units per cubic foot. Three of the animals died early in the experiment and at the autopsy showed no pathologic lung condition. The other 11 were killed at the end of nine months. The lungs showed no indication of pneumonia or tuberculosis but showed an irritated condition in every animal resembling somewhat an asthmatic lung. I feel from the facts stated that emanations may well be an etiological agent causing carcinoma of the lungs.

However, I am inclined to consider that the exposure from drinking activated waters is somewhat different and the ill effects, if any, will appear in the kidneys rather than in the lungs.

REFERENCES

1. SWANN, W. F. G., and DEL ROSARIO, C. The effect of radioactive radiations upon Euglena. *J. Franklin Inst.*, March, 1931, *211*.
2. COLWELL and RUSS. Radium, X-Rays and the Living Cell. Bell and Sons, London, p. 178.
3. RUTHERFORD, ERNST, and others. Radiations from Radioactive Substances. Macmillan, New York, 1930.
4. SONNTAG. Extract from article: Some medical experiences of the Radium Spa Bad Bramback. Published by the administration of the Radium Mineral Spring Co., Bad Bramback i. v.
5. LATTÈS, J. S., and LACASSAGNE, A. Elimination du polonium injecté dans l'organism. *J. de radiol. et d'électrol.*, 1928, *12*, 16–19.
6. Official Gazette Balneology Society of Germany, May, 1932.
7. BEUTEL, A., and WOLDRICH, A. Clinical and roentgenologic study of the development of the Joachimstal bronchial carcinoma. *Ztschr. f. Krebsforsch.*, 1931, *34*, 109–122.
8. LACASSAGNE, A., and LATTÈS, J. Localisations histologiques spéciales du polonium a l'intérieur de certains organes. *Compt. rend. Soc. de biol.*, 1924, *90*, 485–487.
9. Radioactivity and metallo-proteins. *Arch. di sc. biol.*, 1931, *16*, 421–462.

Vol. XXXI, No. 6

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

Editor: Lawrence Reynolds, M.D.
Assistant Editor: Ruth Bigelow, B.S.
Editorial Board: A. C. Christie, M.D. E. H. Skinner, M.D. Lauriston S. Taylor
Advisory Board for Pathology: James Ewing, M.D. Eugene L. Opie, M.D.
Collaborating Editors: The Officers and Committee Members of the Societies of which this Journal is the official organ, whose names appear on this page, are considered collaborating editors of this Journal.
Foreign Collaborators: A. Béclère, M.D., Paris, Gösta Forssell, M.D., Stockholm, G. F. Haenisch, M.D., Hamburg, R. Ledoux-Lebard, M.D., Paris.

Publisher: Charles C Thomas, 220 East Monroe St., Springfield, Illinois

Issued Monthly. Subscription $10.00 per year, $11.00 in Canada and $12.00 in foreign countries. Advertising rates submitted on application: Editorial office, 110 Professional Building, Detroit, Mich. Office of publication, 220 E. Monroe St., Springfield, Ill. Information of interest to all readers will be found on page iv.

Officers and Standing Committees

THE AMERICAN ROENTGEN RAY SOCIETY

President: John T. Murphy, Toledo, Ohio; *President-Elect:* George W. Grier, Pittsburgh, Pa.; *1st Vice-President:* L. R. Sante, St. Louis, Mo.; *2d Vice-President:* H. J. Walton, Baltimore, Md.; *Secretary:* E. P. Pendergrass, University Hospital, Philadelphia, Pa.; *Treasurer:* E. L. Jenkinson, 1439 S. Michigan Ave., Chicago, Ill.; *Historian:* E. H. Skinner, Kansas City, Mo.

Executive Council: John T. Murphy, George W. Grier, L. R. Sante, H. J. Walton, E. P. Pendergrass, E. L. Jenkinson, Lawrence Reynolds, J. Bennett Edwards, B. R. Kirklin, L. Jaches, W. A. Evans, Charles L. Martin, Sherwood Moore, Merrill C. Sosman, Chairman, Peter Bent Brigham Hospital, Boston, Mass.

Committee on Relationship between Radiologists and Hospitals: L. Jaches, Chairman, New York, A. C. Christie, Washington, D. C., W. C. Hill, Cleveland, Ohio.

Committee on Safety and Standards: A. U. Desjardins, Chairman, Rochester, Minn., T. Leucutia, Detroit, Mich., W. D. Coolidge, Schenectady, N.Y., G. E. Richards, Toronto, Ont., F. J. Hodges, Ann Arbor, Mich, H. K. Pancoast, Philadelphia, Pa.

Publication Committee: B. R. Kirklin, Chairman, Rochester, Minn., Ross Golden, New York City, Ralph S. Bromer, Bryn Mawr, Pa.

Finance Committee: J. Bennett Edwards, Chairman, Leonia, N.J., Leslie R. Hess, Hamilton, Ont., Raymond C. Beeler, Indianapolis, Ind.

Advisory Committee to the Moore School X-Ray Laboratory: W. E. Chamberlain, Chairman, Philadelphia, R. R. Newell, San Francisco, E. P. Pendergrass, Philadelphia.

Committee on National Board of Examiners for Radiology: B. R. Kirklin, Rochester, Minn., L. R. Sante, St. Louis, Mo., W. F. Manges, Philadelphia, Pa.

Member, National Research Council: M. C. Sosman, Boston, Mass.

Committee on Arrangements: R. B. Robinson, Chairman, Jenkins Arcade, Pittsburgh, Zoe A. Johnston, Heinz Langer, J. F. McCullough, W. B. Ray, J. W. Fisher, C. S. Caldwell.

Editor: Lawrence Reynolds, 110 Professional Building, Detroit, Mich.

Assistant Editor: Ruth Bigelow, 110 Professional Building, Detroit, Mich.

Editorial Board: A. C. Christie, E. H. Skinner, Lauriston S. Taylor.

Advisory Board for Pathology: James Ewing, Eugene L. Opie.

Thirty-fifth Annual Meeting: Hotel Wm. Penn, Pittsburgh, Pa., Sept. 25–28, 1934.

THE AMERICAN RADIUM SOCIETY

President: Rollin H. Stevens, Detroit, Mich.; *President-Elect:* William H. Cameron, New York City; *1st Vice-President:* George W. Grier, Pittsburgh, Pa.; *2d Vice-President:* John W. Cathcart, El Paso, Texas; *Secretary:* Edward H. Skinner, 1532 Professional Bldg., Kansas City, Mo.; *Treasurer:* Zoe H. Johnston, Jenkins Arcade, Pittsburgh, Pa.

Executive Committee: H. J. Ullmann, Chairman, Santa Barbara, Calif., Sanford Withers, Denver, Colo., E. C. Ernst, St. Louis, Mo.

Program Committee: Wm. H. Cameron, Chairman, New York City, Maurice Lenz, New York City, J. E. Gendreau, Montreal, Que.

Publication Committee: E. H. Skinner, Chairman, Kansas City, Mo., B. F. Schreiner, Buffalo, N.Y., Douglas Quick, New York City.

Research and Standardization Committee: G. Failla, Chairman, New York, H. J. Ullmann, Santa Barbara, Calif., R. B. Greenough, Boston, Mass.

Education and Publicity Committee: G. E. Pfahler, Chairman, Philadelphia, Pa., C. L. Martin, Dallas, Texas, J. S. Ullman, Natchez, Miss.

Committee on National Board of Examiners for Radiology: Henry Schmitz, Chicago, R. H. Stevens, Detroit, H. K. Pancoast, Philadelphia.

19th Annual Meeting: Cleveland, June 11–12, 1934.

EDITORIAL

TRIANGULAR BASAL SHADOWS IN ROENTGENOGRAMS OF THE CHEST

BRONCHIECTASIS is a more common lesion than autopsy reports would seem to indicate. Only about 2 to 4 per cent of the autopsies reported from hospitals call attention to the presence of bronchiectasis, but there is increasing evidence that it is much more prevalent than this. The modern methods of diagnosis, such as the use of the roentgen ray in chest examinations and the introduction of iodized oil into the bronchi, have drawn attention to the widespread prevalence of this disease. Singer and Graham[1] were the first in this country to recognize the importance of the so-called triangular shadows that occur at the bases of the lung as a sign of atelectasis and bronchiectasis of the lower lobe. Their observations were verified during the surgical treatment of their cases but were not studied by means of opaque substances prior to operation. They called attention to the extensive use of lipiodol in France and in South America at that time but they felt that the use of foreign substances in the lung was not wholly necessary to make a diagnosis and that it had other drawbacks such as blocking the smaller bronchi. Richards,[2] in a very comprehensive paper read at the Thirty-third Annual meeting of the American Roentgen Ray Society, again drew attention to the importance of these triangular basal shadows in roentgenograms of the chest, and in an analysis of 2,000 routine chest examinations he encountered this condition ten times. The term triangular basal shadows is, Richards says, a fairly accurate description, since the areas of increased density are usually more or less triangular in outline, occupying the angle formed by the heart and the diaphragm on either side of the vertebral shadows. The outer border of the areas is a straight or slightly curved line extending outwards or downward toward the diaphragm, thus completing the triangle. Modifications of this general descriptive term are occasionally seen. The shadow may be somewhat difficult of demonstration because it is situated behind the heart shadow to a certain extent. Following the injection of lipiodol into the lower main bronchi, it was at once observed that this entire area, namely, the triangular basal shadow area, was composed of atelectatic lung containing numerous bronchiectatic cavities. All of the cases reported by Richards were in adults.

It remained for Anspach,[3] who made a careful study of the triangular basal shadows in infants, to bring out the true nature of the shadow and to demonstrate the successive steps in the production of the common form of bronchiectasis in children, and to show the benefit of certain prophylactic procedures when started early and continued for a long time. He calls attention to the fact that clinical and necropsy observations in relatively advanced cases have been the basis for many theories regarding the development of bronchiectasis but that little has been known about its pathogenesis. No pre-bronchiectatic conditions had been recognized until his report and no serial biopsies had been

[1] Singer, J. J., and Graham, E. A. Roentgen-ray study of bronchiectasis. AM. J. ROENTGENOL. & RAD. THERAPY, 1926, *15*, 54–58.

[2] Richards, G. E. The interpretation of the triangular basal shadows in roentgenograms of the chest. AM. J. ROENTGENOL. & RAD. THERAPY, 1933, *30*, 289–295.

[3] Anspach, W. E. Atelectasis and bronchiectasis in children; a study of fifty cases presenting a triangular shadow at the base of the lung. *Am. J. Dis. Child.*, May, 1934, *47*, 1011–1050.

recorded to show the changes from one period to another. These triangular basal pulmonary shadows that occur in children are an exact counterpart of those seen and reported in adult cases, namely, the mesial border, or altitude, and the inferior border, or base, in the order named, are indistinguishable from the shadows of the spine and leaf of the diaphragm. The well-defined lateral border, or hypotenuse, of the triangle extends from the hilus to a variable point on the diaphragm. The mesial portion of the shadow is partly obscured by the cardiac shadow because the heart, diaphragm and other adjacent structures are drawn toward the involved side. In lateral roentgenograms this density is seen as an isosceles triangle with its apex at the hilus and its base on the diaphragm. Anspach's explanation of these triangular densities differs somewhat from that of the previously published reports. An outstanding feature in the history in the majority of Anspach's cases was that of an acute pulmonary process at the onset, with elevation of temperature which was frequently preceded by a chill. The physical findings approached those of a lobar pneumonia. There was usually an early decrease of breath sounds near the base of the involved side. Moist râles were frequently heard. By the end of forty-eight hours the temperature usually dropped, often suddenly, and from that time on was usually elevated no more than one degree. It was sometimes subnormal. There was a frequent hacking cough or practically no pulmonary symptoms for several weeks. Eventually the cough became moist and expectoration was more frequent, especially after a period of sleep. Roentgenograms of the chest, if made early, showed the triangular shadow to be present but no dilatations of the bronchi were seen. Acute exacerbations of fever were common. Attention is called to the fact that often pulmonary symptoms after the acute onset were so mild that they were overshadowed almost completely by an infection of the upper respiratory tract. Anspach made observations at different periods during the illness of his cases and he was able to study the character of the density of the triangle and to note the fact that this had a direct bearing on the rapidity with which bronchiectasis developed. If the triangular shadow was small and of fluid-like density and remained so, bronchiectasis developed rapidly. If, on the other hand, the triangular basal shadow density disappeared and recurred at frequent intervals, dilatations of the bronchi did not develop or were very slight. If, however, the homogeneous density returned and remained unchanged at subsequent examinations, bronchiectasis developed. If a portion of the triangular density disappeared or if the triangle was small and dense at one examination and then larger and less dense at an early subsequent examination, bronchial dilatations did not occur or were almost negligible in size even after the process had been present for years. These changes were interpreted on the basis of obstruction or drainage of the bronchi. If all of the medium-sized bronchi or the main bronchus to a lobe became occluded, the air distally became absorbed and collapse of the lobe occurred. If one or more bronchi were drained, air entered the lobule or lobules and that portion of the lobe was reinflated and appeared less dense on the roentgenogram. These changes form the basis on which roentgen interpretation of an individual stage must depend and which enable one to estimate the success of treatment and to forecast to some degree the final result.

With rare exceptions, atelectasis accounted for the small triangular area of density in young children in whom the delicate tissues and small caliber of the bronchi formed an ideal situation for an obstruction by tenacious infected secretions.

In an analysis of his cases, Anspach concluded that in all probability the bronchiectasis of the lower lobe develops as follows: "As the bronchi were filled with infected tenacious secretion, the air present distally was absorbed, and atelectasis re-

sulted. If all, or the greater portion of, the lobules of a lower lobe were collapsed, a more or less triangular shadow appeared on the roentgenogram. If complete collapse was maintained sufficiently long because of delayed drainage or because of a constant supply of thick secretion, pathologic changes occurred which weakened the bronchial walls. The pull of the inspiratory muscles immediately brought about an increase in the negative intrathoracic pressure over the normal because of the decreased intrathoracic volume. This intrapleural pull affected the bronchi of the involved lobe because they, through their 'semifluid' filled lumens, were subject to the changes in pressure in the uninvolved portions of the bronchi and trachea and in the pleural space. This difference in pressure tended to bulge or stretch the bronchial wall; the weakest portions subject to the greatest amount of damage (often ulceration) ultimately gave way. When the bronchi were dilated, because of their increased volume and the greater plasticity of thin-walled bronchi expiratory pressure at times was more effectively applied, and some evacuation of the fluid into the main air passages occurred. Air then more frequently entered the alveoli of individual lobules, gradually causing partial reexpansion with corresponding nullification of the abnormally increased intrapulmonary negative pressure. This was a rapid or a slow process, probably depending to some extent on the type of infection present, but mainly on the length of time complete collapse existed. It developed more slowly in older subjects. When all these changes occurred, bronchiectasis existed as usually recognized clinically and roentgenologically in older patients."

From a study of these triangular basal shadows in children and the striking similarity to those reported in the adult, Anspach has been led to suggest that atelectasis is probably a precursor of adult bronchiectasis in many cases. He also calls attention to the part that allergy might play in bringing about the initial obstruction to the bronchi by producing a thick secretion or a slightly swollen lining membrane as observed at necropsy in some of his early cases. The importance of recognizing these triangular basal shadows not only during infancy and childhood but in adult life cannot be too strongly emphasized. If the condition is recognized sufficiently early in infancy and childhood it will save the patient a long drawn out illness, and since the roentgenologist stands in a unique position to demonstrate these changes he should familiarize himself with the appearances of these triangular basal shadows as shown roentgenographically. It is important not only to make a thorough roentgen study of the chest but also to examine the paranasal sinuses, as has been emphasized by Manges and others. Bronchiectasis is all too common and not infrequently the roentgenologist unwittingly contributes to the prolongation of the disease process by not recognizing these triangular basal shadows.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. E. P. Pendergrass, University Hospital, Philadelphia, Pa.
Annual Meeting: Hotel William Penn, Pittsburgh, Pa., Sept. 25–28, 1934.

American College of Radiology
Secretary, Dr. Albert Soiland, 1407 S. Hope St., Los Angeles, Calif.
Annual meeting: Cleveland, 1934.

Section on Radiology, American Medical Association
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio.
Annual meeting: Cleveland, Ohio, June 11–15, 1934.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N.Y.
Annual meeting: Memphis, Tenn., Dec. 3–7, 1934.

Radiological Section, Baltimore Medical Society
Secretary, Dr. W. B. Firor, 1100 N. Charles St., Baltimore.
Meets third Tuesday each month, September to May.

Radiological Section, Los Angeles County Medical Society
Secretary, E. N. Liljedahl, Los Angeles, Calif.
Meets on the third Wednesday of each month at the California Hospital.

Radiological Section, Southern Medical Association
Secretary, Dr. F. B. Bogart, Medical Arts Bldg., Chattanooga, Tenn.
Annual meeting: San Antonio, Texas, November, 1934.

Brooklyn Roentgen Ray Society
Secretary, Dr. A. L. Voltz, 115 Myrtle Ave., Richmond Hill, N.Y.
Meets monthly on first Tuesday, October to April.

Buffalo Radiological Society
Secretary-Treasurer, Dr. Joseph S. Gian-Franceschi, 610 Niagara St., Buffalo, N.Y.
Meets second Monday of each month except during summer months, place of meeting selected by the host.

Chicago Roentgen Society
Secretary, Dr. R. G. Willy, 1138 N. Leavitt.
Meets second Thursday of each month October to May inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. H. G. Reineke, General Hospital, Cincinnati, Ohio.
Meets third Monday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. Otto Glasser, Cleveland Clinic.
Meetings are held at 6:30 P.M. at the Cleveland Chamber of Commerce Club rooms on the fourth Monday of each month from October to April, inclusive.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital.
Meets monthly on first Thursday from October to May, at Wayne Country Medical Society Building.

Florida Radiological Society
Secretary, Dr. W. McL. Shaw, 418 St. James Bldg., Jacksonville, Fla.
Meetings held twice a year, May and November.

Illinois Radiological Society
Secretary, Dr. H. W. Grote, 219 N. Main St., Bloomington, Ill.
Regular meetings held quarterly.

Indiana Roentgen Society
Secretary, Dr. J. N. Collins, Indianapolis, Ind.
Annual meeting each February 22 in Indianapolis.

Michigan Association of Roentgenologists
Secretary, Dr. S. W. Donaldson, St. Joseph's Mercy Hospital, Ann Arbor, Mich. Three meetings a year, Fall, Winter, and Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. S. A. Morton, Columbia Hospital, Milwaukee, Wis.
Meets monthly. Place of meeting designed by the president.

Minnesota Radiological Society
Secretary, Dr. L. G. Rigler, University Hospital, Minneapolis, Minn.

New England Roentgen Ray Society
Secretary, Dr. Thomas R. Healy, 370 Marlboro St., Boston, Mass.
Meets monthly on third Friday, Boston Medical Library.

New York Roentgen Society
Secretary, Dr. C. W. Schwartz, 33 E. 68th St., New York.
Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Roentgen Ray Society
Secretary, Dr. Major Fleming, Rocky Mount, N.C.
Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

Central New York Roentgen Ray Society
Secretary, Dr. F. C. Rulison, Medical Arts Bldg., Syracuse, N.Y.
Three meetings a year—January, May, and November.

Pacific Roentgen Club
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif.
Meets annually, during meeting of California Medical Association

Pennsylvania Radiological Society
Secretary, Dr. W. E. Reiley, Clearfield, Pa.

Philadelphia Roentgen Ray Society
Secretary, Dr. Thomas Loughery, Germantown Hospital.
Meeting first Thursday of each month from October to May inclusive, at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Rochester Roentgen Ray Society, Rochester, N.Y.
Secretary, Dr. W. W. Fray, Strong Memorial Hospital.
Meets monthly on second Thursday from October to May, inclusive, at 7:45 at the Rochester Medical Association Building.

St. Louis Roentgen Club
Secretary, Dr. W. K. Mueller, University Club Bldg.
Meets first week of each month. Time and place of meetings designated by president.

South Carolina X-Ray Society
Secretary, Dr. R. B. Taft, 105 Rutledge Ave., Charleston.
Meets at time and place of South Carolina State Medical Association.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

Tennessee Radiological Society
Secretary, Dr. F. B. Bogart, 311 Medical Arts Bldg., Chattanooga, Tenn.
Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary-Treasurer, Dr. J. H. Smith, Lubbock, Texas.
Meets annually one day preceding the meeting of the Texas State Medical Association. Next Meeting. San Antonio, May 7, 1934.

University of Michigan Department of Roentgenology Staff Meeting.
Secretary, Dr. C. B. Peirce, University Hospital, Ann Arbor, Mich.
Meets each Monday evening from September to June, at 7 P. M. at University Hospital.

University of Wisconsin Roentgen Club
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis.
Meets monthly on last Thursday, October to April, Service Memorial Institute, at 4:30 P.M.

Virginia Roentgen Ray Club
Secretary, Dr. V. W. Archer, University Hospital, University, Va.
Meets annually in October.

Cuba

Sociedad Cubana de Radiologia y Fisioterapia
Secretary, Dr. Luis Fariñas, Animas 110, Havana, Cuba.
Meets monthly in Havana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Meets on the third Thursday of each month, from November to June inclusive, at 8:15 P.M., at 32 Welbeck St., London, W. 1., or as advertised.

Electro-Therapeutic Section of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month during the winter at 8:30 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Colin Macdonald, Lister House, 61 Collins St., Melbourne, Australia.
Meets monthly at Melbourne during the winter.

Section on Radiology, Canadian Medical Association
Secretary, Dr. A. H. Rolph, 160 St. George St., Toronto, Ont.

Indian Radiological Association
Secretary, Sd. Subodh Mitra, 148 Russa Rd., Calcutta.
Meets annually in January, and at such places and times as the Council may appoint.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. P. C. Fenwick, The Hospital, Christchurch. Meets annually.

Continental Europe

Belgian Society of Roentgenology
Secretary, Dr. J. Boine, Avenue des Allies, 134, Louvain, (Belgian).
Meets monthly on second Sunday at d'Egmonds Palace, Brussels, except in the summer time.

Société de Radiologie Médicale de France
Meets monthly on second Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean, La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse, Biel.
Meets annually in different cities.

Société Française d'Electrothérapie et de Radiologie Médicale
Meets monthly on fourth Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

Association of German Roentgenologists and Radiologists in Czecho-Slovakia
Secretary, Dr. Walter Altschul, German University, Prague, II/52.

Deutsche Röntgen-Gesellschaft (Gesellschaft für Röntgenkunde und Strahlenforschung)
Meets annually in April in different German cities, at least once in five years in Berlin. Meets in addition every two years with the Gesellschaft deutscher Naturforscher und Aerzte.
Permanent Secretary, Professor Dr. Haenisch, Klopstockstrasse 10, Hamburg, Germany.

Dutch Society of Electrology and Roentgenology
Holds two meetings a year in Amsterdam, one in the Spring, and one in the Fall.

Societa Italiana Radiologia Medica
Secretary, Professor M. Ponzio, University of Turin, Turin.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Maracine 30, Bucharest, Rumania.
Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad, USSR, in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson.
Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin.
Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky.
Meets monthly on the first Monday at 8 o'clock, the place of meeting being selected by the Society.

Polish Society of Radiology
Secretary, Dr. Jan. Kochanowski, 45 Gornoslazka St., Warsaw.
Meets annually.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. B. Krynski, 11 Zielna St.
Meets once a month except in the summer time.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association. Each of the following societies, with exception of the Denmark Society, meets every second month except in the summer time.

Society of Medical Radiology of Sweden
Meets in Stockholm.

Society of Medical Radiology in Norway
Meets in Oslo.

Society of Medical Radiology in Denmark
Secretary, Dr. H. Scheuermann, Copenhagen.
Meets on the second Wednesday of each month from October to July in Copenhagen, at 8 o'clock in the State Institute of Roentgenology.

Society of Medical Radiology in Finland
Meets in Helsingfors.

Vienna Society of Roentgenology
Meets first Tuesday each month, October to July.

Orient

Japan X-Ray Association
c/o Orthopedic Surgery, Tokyo Imperial University
Meets annually in April.

Kinki Roentgen-Abend Society
Director, Dr. Prof. Taiga Saito, Ogawaoike, Kyoto, Japan.
Meets bi-monthly on third Sunday.

MADRAS GOVERNMENT INSTITUTE OF RADIOLOGY

One of the inexplicable signs of the times is the vast amount of space given over by the daily press to what many people consider inconsequentials; for instance, if a certain political propagandist chooses to change his seat from the shady side to the sunny side of the street and to drink goat's milk this week and to refrain from drinking it next week, or if a certain premier chooses to announce that he will increase his land forces or his cruisers to a certain extent or that another will increase the fortifications along the boundaries, these happenings are heralded over the radio and in the news as matters of great moment. But happenings and incidences that are of far greater importance to the well-being of a country, such as the opening of an institute of radiology, must await the slower transport of ship and train.

Such an announcement has recently been received of the opening of the Madras Government Institute of Radiology, Madras, India. The Madras Government General Hospital has existed since 1664 when the "Council at Fort St. George wrote to the Agent, Sir Edward Winter, . . . that 'fresh souldiers which came forth this year, takeing up their habitation in the bleake winde in the hall, fell sick. Four of them are dead, about tenn remaine at this time very sick. . . . Soe, rather than to see Englishmen dropp away like doggs in that

Fig. 1. Government Institute of Radiology, Madras, India.

Fig. 2. Façade of the Government Institute of Radiology.

Manner for want of Christian charity towards them, we have thought it very convenient that they might have an house on purpose for them, and people appointed to looke after them and to see that nothing comes into them, neither of meat nor drinke, but what the Doctor alloweth.' Thus started the first British Hospital in Madras. The house taken up was that of Andrew Cogan, who became the Company's Agent in 1639, and who, with Francis Day, founded the Fort; the rent was two pagodas a month."

During these three centuries the Madras General Hospital has contributed to the welfare of the sick of India and each year throughout its long service it has seen the Hospital grow from a small institution to the present splendid position it holds in the medical world today. It is of particular importance to roentgenology that in the recent scheme for the enlargement of this institution they made provision for an Institute of Radiology. While we in this country are discussing the future of radiology, a few determined, far-sighted individuals in India have placed the future of radiology in that country on a solid basis as instanced by the Madras Institute of Radiology. The fulfillment of this dream can be attributed to Captain T. W. Barnard who is the head of the Institute, and the foreword to the brochure announcing the Madras Institute of Radiology states, "Captain Barnard, as the first Director, is entitled to our best wishes for the success of his ambitious experiment and the Madras Presi-

dency will be proud to be in the vanguard of radiological practice and research."

This new Institute of Radiology was opened on March 26 in the presence of His Excellency, the Governor of Madras and Lady Beatrix Stanley, and other distinguished visitors. The accompanying illustrations give some idea of the extent and scope of the buildings and the following is a description of the Institute taken from *The Hindu*, Monday, March 26, 1934.

The new Radiological Institute when completely equipped will, it is expected, to be the best of its kind in India and in the East.

X-Ray and electric treatment apparatus has been in use in this Presidency for many years now, the first outfit for the General Hospital having been obtained in or about the year 1900, within five years of the discovery of the ray. Since then the equipment in the hospital has made rapid progress and what was once a primitive type of apparatus run from a small battery of accumulators has now developed into one of the most up-to-date Radiological centres in the world, constructed at a cost of about Rs. 8 lakhs.

Since its inception, the Institute has gone through many changes. In 1920, the Government appointed Capt. T. W. Barnard to organise the Radiological Service which was to include the building of a teaching centre for South India. The attendance at the institute in 1920 was 3,465 and in two years it nearly quadrupled itself and in 1933 it had advanced to 75,780. The rapid increase in the number of patients treated has made the present building a necessity.

The block of buildings in which the institute will be housed is a fine adjunct to the Hospital of two storeys and capable of further being built upon. The two blocks of the building are directly connected with the surgical block of the Hospital. On the ground floor are situated the Radio Transformer rooms, a large demonstration hall and the museum, Radiographic and screening room for examining patients in, equipment of Dean's apparatus, emergency X-Ray and dental X-Ray departments and the Radiographic department equipped with Victor KX5 apparatus and accessories. Between each pair of rooms is a large dark room with connecting revolving light proof cup-boards enabling the transfer of films from the X-Ray department to the dark room. Accommodation is also provided on this floor for a bed-sitting room for patients during Barium Meal examinations, a store room and a room for air-conditioning. In another part of the ground-floor will be located the large remedial exercises and massage hall, clinical photographic and electrocardiographic departments with necessary dark rooms, the X-Ray apparatus being controlled from the "operators' corridor" outside.

On the first floor is the hall providing accommodation for electrical and heat treatments, for the Urological and Surgical Diathermy theatre, the high tension wire for the former being brought through the floor from the Radiographic department below. At the other end is located the Hydro-Therapy section. The Ultra Violet Radio centre, a Radium theatre, Radon laboratory and a Radium Safe Room are also accommodated on this floor.

The X-Ray and Radium treatment rooms, it is stated, have been purposely built on the southern side facing the open military grounds and the Cooum to avoid possible radiation dangers to the other departments. Care has also been taken to afford protection to the walls by incorporation of Barium Sulphate in the building bricks. Sample sections of the walls were tested in Madras and then forwarded to London, where the National Physical Laboratory confirmed the results obtained here.

The Institute, it is claimed, is well equipped for becoming an excellent training centre for South India, possibly for all India. In this connection it is stated that the University of Madras may grant Diplomas in Radiology in the near future. The authorities also hope that the Institute will become self-supporting from the fees which will be levied from all patients who could afford to pay. Details of syllabus and other arrangements for teaching are nearing completion and it is expected that examinations for diploma will be held from 1935.

FOURTH INTERNATIONAL CONGRESS OF RADIOLOGY

Attention is again called to the fact that the Fourth International Congress of Radiology will be held at Zurich and St. Moritz, Switzerland, from July 24 to 31, 1934, under the patronage of the President of the Swiss Confederation, Dr. Marcel

Pilet-Golaz. All of the leading nations of the world will send delegations. There will be meetings in the University and the Federal Institute of Technology at Zurich from July 24 to 28. A delegates luncheon will be given on the opening day, with the Swiss Roentgen Society acting as host, also a reception and ball. A Festival of Swiss National Costumes is to be held in honor of the visitors on July 27. The trip to St. Moritz, which will provide much scenic entertainment, is scheduled for July 28, on which day a reception will be tendered to the delegates by this famous Engadine resort. A final banquet will be held on the closing day.

AMERICAN ROENTGEN RAY SOCIETY

ANNUAL MEETING

The Thirty-fifth Annual Meeting of the American Roentgen Ray Society will be held at Pittsburgh, Pennsylvania, on September 25 to 28, 1934. The scientific sessions will be held at the William Penn Hotel where adequate provision has also been made for the Scientific and Commercial Exhibits. Dr. G. W. Grier, the President-elect, who has charge of the program and arrangements for the meeting has an interesting program well under way, the preliminary report of which will be published in the August issue of the Journal. The railroads have granted a reduction in fares, a special notice of which will be found below. The Scientific Exhibit is in charge of a committee headed by Dr. B. R. Kirklin. The members of the Society who have interesting films to exhibit should immediately communicate with Dr. Kirklin; fortunately the space selected for these exhibits is ample to display them in the proper manner. As is usual at the meetings, the manufacturers will display their latest products, and all in all the Pittsburgh meeting is one that the members of the Society can ill afford to miss.

REDUCED RAILROAD FARES TO THE MEETING

The railroads have allowed the members of the American Roentgen Ray Society and their families attending the meeting in Pittsburgh a reduced fare of fare and one-third of the current one way first class fares on the certificate plan, provided 100 or more certificates are presented. The certificates are on sale September 19 to 25, 1934, and the final honoring date is October 2. Members attending the meeting are urged to secure these certificates.

SUBJECT INDEX TO VOLUME XXXI

(abs) = abstract (E) = editorial

ABDOMEN, study of hydro-aerial images of (abs), 415
ABRODIL, used in pyelography (abs), 122; (abs), 123; (abs), 124
ABSCESS, bone, unusual migration of, originating in vertebra (abs), 138
lung, putrid, bronchopulmonary segment with special reference to, 328
perigastric, complicating peptic ulcer, pathogenesis of (abs), 420
subdiaphragmatic, 177
ACETABULUM, intrapelvic protrusion of, 346
ACID-BASE BALANCE in genital carcinoma in women (abs), 282
ACTINOMYCOSIS, roentgen therapy of, 823
ADDISON'S DISEASE, calcification of suprarenal glands in (E), 93
roentgen examination, 394; (abs), 420
ADENOMAS, basophil, of pituitary gland and their clinical manifestations (abs), 408
ADRENALIN, liver retraction test with (abs), 417
AINHUM, roentgen findings, 349
ALVEOLI, inverse ratio in roentgen visualization of, after injection of contrast media, 323
AMERICAN BOARD OF RADIOLOGY, 258
AMERICAN COLLEGE OF RADIOLOGY, relation of, to future of radiology, 164
AMERICAN RADIUM SOCIETY, program of Nineteenth Annual Meeting, 684
AMERICAN ROENTGEN RAY SOCIETY, annual meeting, 845
reduced railroad fares to, 845
scientific exhibit, 395
ANEMIA, erythroblastic (abs), 571
racial and familial, of children (abs), 571
ANGIOMAS, treatment by radium therapy (abs), 573
Annals of Surgery celebrates Golden Jubilee, 262
"ANNULAR SHADOWS," significance of, in roentgenograms of chest (abs), 410
ANTIPERISTALSIS of colon (abs), 118
AORTA, coarctation of (abs), 558
mesaortic descending, abnormal situation (abs), 698
right arching of (abs), 414
APOPHYSES, persistent, of vertebral column (abs), 702
APPARATUS, advances in x-ray engineering with reference to high potentials (abs), 576
device for taking pulmonary roentgenograms under control of heart beat (abs), 143
focal spot camera, 399
for measuring density of roentgen films, 550
frame for holding infants upright during roentgenography, 396
improved device for synchronizing roentgenographic exposure with patient's pulse, 104
"light-pencil" for marking on roentgenographic and photographic emulsions, 554
new aiming instrument to make focusing easier in gynecological deep irradiation (abs), 719
new roentgenoscopic and roentgenographic grid (abs), 287
overhead aerial high tension system, 690
roentgen tube with internal radiation protection for 400 kv. operation voltage (abs), 577
simple radium detector, 689
valve tube for 400 kv. tension (abs), 577
wet-film viewing box, 398
APPENDICITIS, chronic, results and limitations of roentgen diagnosis (abs), 564
chronic, roentgen examination of inferior quadrant (abs), 564
chronic, solution of roentgen diagnostic problem in, 792
APPENDIX, pathological, 340
roentgen study of active movements of (abs), 118
roentgen visualization of (abs), 573
ARTERITIS, treatment by radiotherapy (abs), 431
obliterans, treated by roentgen therapy of suprarenal region (abs), 576
ARTHRITIS, certain cases of, associated with parathyroidism (abs), 708
costovertebral (abs), 136
relation of intestinal tract and diet to treatment of (abs), 137
use of parathyroidectomy for (abs), 708
ASCARIS, roentgen study of lungs of children with (abs), 412
ASTHMA, roentgen therapy of pancreas in treatment of (abs), 576
ASTRAGALUS, dislocation of (abs), 709

BACKACHE, lumbosacral, and sciatic pain, oblique view for demonstration of articular facets in, 173
BASEDOW'S DISEASE: See GOITER, EXOPHTHALMIC
BASOPHILISM, pituitary (abs), 408
BILHARZIASIS, urinary, roentgen manifestations, and calculous formations in Egypt, and intravenous pyelography, 208
BLADDER, closing mechanism of (abs), 120
diverticulum of (abs), 122
"BLEEDING NIPPLE," new method of treatment by radium implantation, 819
BLOOD, alterations in pH of, in cancer following roentgen and gamma irradiation, 532
changes in picture of, after deep roentgen irradiation, and their significance in prognosis (abs), 579

corpuscles, red, sedimentation reaction of, immediately before and after roentgen irradiation (abs), 281
diseases of, roentgen therapy (abs), 141
diseases of, severe, effect of very small and larger doses of roentgen rays in (abs), 276
disturbances in circulation of, radium irradiation of blood vessels in (abs), 277
vessels, radium irradiation of, in circulatory disturbances (abs), 277
Bone, and joint changes in hemophilia, 487
marrow, action of roentgen rays on (abs), 144
marrow, histological studies of, in rabbits following intravenous injection of thorium dioxide, 512
tuberculosis, two peculiar cases of (abs), 135
Bones, and ligaments, massive fluorosis of (abs), 125
changes in, in hemangiosarcoma of spleen (abs), 134
long, non-rachitic cupping of, in new-born (abs), 136
Book Reviews
The 1933 Year Book of Radiology. Edited by Charles A. Waters and Ira I. Kaplan, 100
Functional Disorders of the Large Intestine and their Treatment. By Jacob Buckstein, 100
Diagnostico de la Apendicitis cronica. By F. Gallart Monés and T. A. Pinós, 101
Precis d'eléctro-radiologie appliquée a l'odontostomatologie. By E. Pierron, 101
Human Cancer: Etiological factors; precancerous lesions; growth; spread; symptoms; diagnosis; prognosis; principles of treatment. By Arthur Purdy Stout, 263
Nasal Accessory Sinuses. By Frederick M. Law, 263
Grundlagen und Praxis der Röntgenstrahlendosierung: Dosismessung und Dosisfestsetzung. By H. Holthusen, 264
Radiodiagnostico en la Infancia. By Manual and Francisco Arce, 265
Manuel de radiodiagnostic clinique. Volumes I and II. By R. Ledoux-Lebard, 549
Compendio di radiologia medica (radiodiagnostica e radioterapie) ad uso degli studenti e dei medici pratici. By G. B. Cardinale and Mario Maino, 549
The Science of Radiology. Edited by Otto Glasser, 686
Trattato di Roentgen- e di Curie-terapia. Edited by Felice Perussia and Enzo Pugno-Vanoni, 686
Neurology. By Roy R. Grinker, 687
Borderline rays in ophthalmology (abs), 573
Bordier method in treatment of acute anterior poliomyelitis (abs), 575
Brain, meningeal fibroblastomas on under surface of temporal lobe and their surgical treatment (abs), 695
Breast, advanced cancer, radiotherapy and surgery (abs), 280
cancer, prophylactic irradiation (abs), 575
carcinoma, roentgen irradiation in treatment of, 46
new method for treatment of "bleeding nipple" by radium implantation, 819
Bronchi, inverse ratio in roentgen visualization of, after injection of contrast media, 323
Bronchography, an essential and safe adjunct in study of pulmonary tuberculosis, 301
in amplification of roentgen film of chronic pulmonary tuberculosis, 289
in relation to pathology of pulmonary tuberculosis, 308
Bronchopulmonary segment with special reference to putrid lung abscess, 328
Bronchus, primary carcinoma, diagnosis and treatment, 145

Calcification, extensive, roentgen evidence of, in osteitis fibrosa cystica, 593
in tuberculosis of suprarenal glands, roentgen study in Addison's disease (abs), 420
intracardiac (E), 678
intracardiac, roentgen diagnosis, 1
myocardial, 766
of suprarenal glands in Addison's disease (E), 93
pathological (abs), 429
subcutaneous, of phlebolithic or phlebosclerotic character in leg (abs), 138
Caldwell Lecture (1933), 145
Camera, focal spot, 399
Cancer, alterations of pH of blood in, following roentgen and gamma irradiation, 532
buccal, lingual and facial, treatment (abs), 574
effect of radium and bismuth on (abs), 283
of breast, advanced, radiotherapy and surgery in (abs), 280
of breast and uterus, osseous metastases from (abs), 566
of breast, prophylactic irradiation (abs), 575
of cervix, results of operation and irradiation (abs), 719
of female genitalia, methods and results of treatment (abs), 141
of mouth, management of, and cervical lymphatics, 366
operative and radiotherapeutic treatment (abs), 279
relation of radiation to genetics and mutation theory of cancer (E), 252
ulcers, red fluorescence in (abs), 282
Carcinoma, bronchiogenic, treatment (E), 544
genital, in women, acid-base balance (abs), 282
in chronic osteomyelitis, 787
incidence in certain chronic ulcerating lesions of stomach (abs), 419
mammary, roentgen irradiation in treatment, 46

of cervix and vagina, statistics of, for years 1923–1925 (abs), 142
of cervix, problems in radiation therapy of, 60
of cervix, treatment (abs), 281
of tonsils, review of 122 cases (abs), 717
of vulva, results of irradiation (abs), 142
ovarian, causing secondary infiltrating stenosis of sigmoid colon (abs), 421
primary, of bronchus or lung, diagnosis and treatment, 145
primary, of duodenum, 204
roentgen, cicatrized by radium (abs), 574
CARDIOVASCULAR DISEASE, Paget's osteitis deformans in relation to (abs), 704
CAROTID SINUS, treatment of vascular hypertension by roentgen excitation of (abs), 578
CECOCOLIC SPHINCTER, spasm of (abs), 700
CECUM, tumor of, simulating ileocecocolic invagination (abs), 116
CELLS, living, action of radiations of short wave length on (abs), 286
CELLULOID as support in application of radium (abs), 283
CERVICAL RIBS, relation of congenital deformities of hand to (abs), 135
CHEST, in children, cystic appearances (abs), 411
roentgen examination of, in lateral decubitus, 41
roentgenograms of, taken with breath held in expiration, 266
significance of "annular shadows" in roentgenograms of (abs), 410
triangular basal shadows in roentgenograms of (E), 838
CHILDHOOD, pulmonary tuberculosis, 16
CHILDREN, cystic appearances in chests of (abs), 411
disease of gallbladder in (abs), 558
examination of, whose mothers have been irradiated (abs), 713
intestinal stasis in (abs), 421
lungs of, with ascaris, roentgen study (abs), 412
roentgen findings in lead poisoning in (abs), 712
tarsal scaphoiditis in (abs), 427
tuberculous pulmonary infiltrations in (abs), 411
CHLOROMA, aleukemic, myelogenous (abs), 139
CHOLECYSTITIS, gangrenous gaseous, roentgen diagnosis (abs), 418
CHOLECYSTOGRAPHY, applications of syncholia and synuria of certain iodized compounds (abs), 430
by oral route and its value (abs), 416
importance of, in diagnosis of gallbladder disease (abs), 115
in cases of impaired liver function (abs), 415
interpretation of (abs), 115
oral, 227
rapid (abs), 415
two new techniques—method of Sandström and of Antonucci (abs), 417
COARCTATION of aorta (abs), 558
COLLOID SUSPENSION, roentgen examination of large intestine by impregnation with thin layer of (abs), 118
COLON, antiperistalsis of (abs), 118
changes of form and position (abs), 565
ectopy of (abs), 565
normal and pathologic movements, roentgen observations (abs), 421
roentgen diagnosis of diseases of, 607
roentgen signs of diverticulosis and diverticulitis of (abs), 423
sigmoid, secondary infiltrating stenosis in ovarian carcinoma (abs), 421
studies on; variations in fixation of cecocolon, 751
treatment of megacolon by parathormone (abs), 421
wall changes and stenoses of, as result of extracolic disease (abs), 421
CORONARY DISEASE, roentgen diagnosis, 588
CUNEIFORM BONE, internal, osteochondritis of (abs), 711
single, dislocation of (abs), 710
CYSTS, intrathoracic, in children (abs), 411

DEOSSIFICATION, parathyroid, roentgen studies, 496
DERMATOLOGY, question of roentgen injury in (abs), 275
DIAPHRAGM, adhesions and folds of (abs), 698
defects of, seen in roentgenogram (abs), 409
DIVERTICULA, multiple, of jejunum and duodenum, 200
of pericardium (abs), 413
pointed, of fundus of stomach (abs), 700
DIVERTICULITIS of colon, roentgen signs (abs), 423
DIVERTICULOSIS of colon, roentgen signs (abs), 423
DIVERTICULUM of bladder (abs), 122
of epiglottic valleculae (abs), 409
of gallbladder, 603
Zenker's (abs), 409
DOSAGE, determination of, for long radium or radon needles, 74
variations in, dependent on wave length, 538
DOSIMETRY, chemical (abs), 578
Hammer ionometer and relation between R (Solomon) and r (international) unit (abs), 577
DUODENITIS and its roentgenologic characteristics, 581
DUODENUM, chronic intermittent obstruction of (abs), 419
multiple diverticula of, 200
primary carcinoma of, 204
stasis of, in icterus catarrhalis (abs), 561
typical variation in position of—duodenum inversum (abs), 699
DWARFISM, renal, and rickets (abs), 424
DYSCHONDROPLASIA FOETALIS (abs), 424
DYSHIDROSIS, new indirect roentgen method for treatment of (abs), 140
DYSTROPHY, cystic, sixteen observations (abs), 426

Echinococcus cyst attached to kidney, 541
Ectopy of colon (abs), 565
Eczema, new indirect roentgen method for treatment of (abs), 140
Editorials
Calcification of the suprarenal glands in Addison's disease, 93
Burton J. Lee, 95
Resolution of the New York Roentgen Society, 97
The relation of radiation to genetics and the mutation theory of cancer, 252
Radiologic service in the United States, 387
The treatment of bronchiogenic carcinoma, 544
Intracardiac calcification, 678
Stanley Melville, 680
Triangular basal shadows in roentgenograms of the chest, 838
Effect of radiations of different wave length on transplantable mouse sarcoma No. 180, 614
of radiations of short wave length on living cells (abs), 286
of radium with bismuth on cancer and growing tumors (abs), 283; (abs), 284
of roentgen irradiation on mineral metabolism (abs), 580
of roentgen rays on bone marrow (abs), 144
of very small and larger doses of roentgen rays in severe blood diseases (abs), 276
stimulant, of roentgen rays on normal tissue (abs), 580
Elbow, radial exostosis complicating anterior dislocation of (abs), 567
Electron emission, early history, 244
Emulsions, roentgenographic and photographic, "light-pencil" for marking on, 554
Encephalography, differential diagnosis by (abs), 696
improved method (abs), 696
under nitrous oxide anesthesia, 784
Enchondromas, calcified, roentgen appearance (abs), 709
Epituberculosis, 721
Epulis, treated with radium (abs), 573
Erysipelas, roentgen therapy of (abs), 277
Esophagus, pouches of (abs), 409
roentgen diagnosis of varices of (abs), 409
tertiary syphilis of, 773
Estrus as sign of beginning of ovulation after temporary roentgen sterilization (abs), 715
Exostosis, radial, complicating anterior dislocation of elbow (abs), 567
regression of, under roentgen irradiation (abs), 715

Femur, lesser trochanter of, some unusual changes in, 224
ununited intracapsular fractures of neck of, roentgenologically considered, 433
Fibroblastomas, meningeal, on under surface of temporal lobe and surgical treatment (abs), 695
Fibromas, nasopharyngeal, treatment by some physical agents (abs), 574
Fibromyoma of uterus, irradiation in treatment of, 51
Fingers, congenital stiffness (abs), 424
Fistula from duodenum into common duct (abs), 116
post-traumatic cerebrospinal fluid, demonstrated roentgenologically (abs), 408
spontaneous internal biliary, cases of (abs), 115
spontaneous internal biliary, roentgen demonstration, 31
Fluorescence, red, in cancer ulcers (abs), 282
Fluorosis, massive, of bones and ligaments (abs), 125
Foot, congenital fusion of bones of (abs), 570
"fork strain" of (abs), 428
Fracture of capitellum and trochlea, combined with fracture of external humeral condyle (abs), 709
of second metatarsal (abs), 428
transverse, of sacrum, 676
Fractures, marching, of metatarsal bones (abs), 712
intra-articular, of capitellum and trochlea of humerus (abs), 423
of inferior extremity of radius, reduction under the screen (abs), 423
of spine, results (abs), 425
old, action of radiotherapy of suprarenal region on constitution of osseous callus in (abs), 432
treatment of, by skeletal devices (abs), 423
ununited intracapsular, of femoral neck roentgenologically considered, 433

Gallbladder, disease of, in children (abs), 558
diverticulum of, 603
importance of cholecystography in diagnosis of disease of (abs), 115
roentgen tests of function of (abs), 699
studies of function of; study of alleged impediment in cystic duct to passage of fluids (abs), 698
Gallstones, roentgen appearance and chemical composition (abs), 699
Ganglioneuroma (abs), 565
Gangrene, diabetic, radiotherapy of suprarenal regions in (abs), 575
Gastroenterology, unification of opaque meal in (abs), 559
Gastrointestinal tract, changes of, during course of uremia (abs), 119
complete examination of, in roentgen practice (abs), 559
disturbance of, following left-sided phrenicoexairesis (abs), 119
relation of, and diet to treatment of arthritis (abs), 137

syphilitic stenoses in (abs), 118
GASTROSCOPIC STUDY of gastric ulcer (abs), 560
GERMAN ROENTGENOLOGISTS and Radiologists in Czechoslovakian Republic, report of 1933 meeting, 393
GOITER, exophthalmic, treatment of, by roentgen therapy of suprarenal region (abs), 576
exophthalmic, treatment of, with radium and results (abs), 716
GRANULOMATOSIS, MALIGNANT: See Hodgkin's disease
GRANULOPENIA (abs), 428
GYNECOLOGICAL CONDITIONS, influence of, on genitourinary tract shown by simultaneous injections of skiodan or uroselectan and lipiodol (abs), 701
new aiming instrument to make focusing easier in deep irradiation in (abs), 719

HAND, congenital deformities, relation to cervical ribs (abs), 135
HEART, anteroposterior roentgen appearance of, as basis for estimating enlargement of particular cardiac region (abs), 698
congenital disease of, in infants, 470
differential diagnosis of shadows within cardiac silhouette (abs), 413
in funnel chest (abs), 414
intracardiac calcification (E), 678
position of right auricle and right ventricle in erect human subject (abs), 413
right aortic arch (abs), 414
roentgen diagnosis of intracardiac calcification, 1
roentgen findings in tricuspid disease (abs), 413
shadow of, densities in (abs), 698
study of, by roentgen kymography (abs), 697
tumors of (abs), 413
HEMANGIOSARCOMA of spleen, changes in bones (abs), 134
HEMOPHILIA, bone and joint changes in, 487
roentgen ray as aid in diagnosis of, 597
HIP JOINT, diagnosis of diseases of (abs), 135
HISTORY, early, of electron emission, 244
HODGKIN'S DISEASE, prognosis of, in connection with 80 cases (abs), 431
radiotherapy of (abs), 430
with cavity formation in the lung (abs), 410
HUMERUS, intra-articular fractures of capitellum and trochlea of (abs), 423
HYDRONEPHROSIS, retrograde ureteropyelography in (abs), 122
HYPERCALCEMIA, acute (abs), 429
HYPERPARATHYROIDISM, a distinct disease entity (abs), 706
HYPERTENSION, vascular, treatment by roentgen excitation of carotid sinus (abs), 578

ICTERUS CATARRHALIS, duodenal stasis in (abs), 561
ILEUM, tumor of, causing recurrent invagination (abs), 116
ILIUM, paraglenoid sulci of (abs), 569
INDIANA ROENTGEN SOCIETY, 548
INFANT, spontaneous pneumothorax in, 770
INFANTS, congenital cardiac disease in, 470
frame for holding infants upright during roentgenography, 396
INJURY, roentgen, question of, in dermatology (abs), 275
INJURIES caused by roentgen rays and radium, cases of (abs), 574
INSTITUTE OF RADIOLOGY of Madras Government, 843
INTERNATIONAL CONGRESS OF ELECTRORADIOBIOLOGY, 548
INTERNATIONAL CONGRESS OF RADIOLOGY, FOURTH, 260, 395, 844
rules and regulations, 260
INTERVERTEBRAL DISC, calcareous deposits in (abs), 703
normal and pathological physiology of nucleus pulposus of (abs), 568
INTESTINE, acute obstruction, roentgen diagnosis (abs), 117
large, roentgen examination of, by impregnation with thin layer of colloid suspension (abs), 118
large, surgical treatment of tuberculosis of (abs), 700
polyposis of (abs), 422
roentgen study of invagination of (abs), 117
small, roentgen diagnosis of invagination of (abs), 116
small, roentgen diagnosis of stenosis of (abs), 562
small, roentgen diagnosis of tumors of (abs), 420
small, roentgen study of last loop of (abs), 563
small, roentgen study of topographical and morphological changes in, on changing from erect to supine position (abs), 117
small, tuberculoma of (abs), 563
stasis of, in infants and children (abs), 421
INVAGINATION, chronic ileocecocolic, simulated by tumor of cecum (abs), 116
intestinal roentgen, study (abs), 117
of small intestine, roentgen diagnosis (abs), 116
recurrent, from tumor of ileum (abs), 116
IODIZED OILS, in myelography, dangers, inconveniences and injuries (abs), 565
IRRADIATION, protracted, biological principles of (abs), 140
protracted fractional, of malignant tumors (abs), 144
roentgen, as nonspecific stimulant therapy (abs), 276
roentgen, pachymeningitis after? (abs), 275

JANEWAY MEMORIAL LECTURE (1933), 153
JEJUNUM, multiple diverticula, 200

KELOID, radiation therapy of, and keloidal scars, 238

Kidney, echinococcus cust attached to, 541
Kidneys, retrograde ureteropyelography in hydronephrosis (abs), 122
roentgen evidence of extensive calcification of, in osteitis fibrosa cystica, 593
scoliosis caused by disease of (abs), 120
Kymography, roentgen, study of heart by (abs), 697
Kyphosis of old age (abs), 427

Lantern slides, filing of, 110
Lead poisoning in children, roentgen findings (abs), 712
Lee, Burton J., obituary notice (E), 95
portrait, 95
resolution of New York Roentgen Ray Society, 97
Leg, subcutaneous calcifications of phlebolithic or phlebosclerotic character in leg (abs), 138
varicose disease, roentgen observations (abs), 138
Leucemia, case of, in radium worker (abs), 283
chronic, results of roentgen treatment (abs), 279
chronic myelogenous, treated by radiation (abs), 572
chronic myeloid, treatment (abs), 572
"Light-pencil" for marking on roentgenographic and photographic emulsions, 554
Lipoids, pneumonia due to aspiration of (abs), 412
Liver, cholecystography in cases of impaired function of (abs), 415
histological studies of, in rabbits following intravenous injection of thorium dioxide, 512
method of roentgen exploration of, in lithiasis and lithiatic icterus (abs), 418
retraction test of adrenalin (abs), 417
Lung, putrid abscess of, bronchopulmonary segment with special reference to, 328
Lungs, accessory lower lobe, roentgen appearance (abs), 697
acute massive collapse, 547
bone formation in (abs), 697
capillary lines of (abs), 558
cavity formation from Hodgkin's disease (abs), 410
historical review of question of roentgen demonstration of cavities caused by destruction of lungs in early tuberculosis (abs), 697
inverse ratio in roentgen visualization of bronchi and alveoli after injection of contrast media, 323
localized roentgen opacities in region of interlobar fissures of (abs), 412
of children with ascaris, roentgen study (abs), 412
posterior lobe (abs), 697
primary carcinoma, diagnosis and treatment, 145
specificity of consolidation of, in tuberculous patients, 721
syphilis of (abs), 410
syphilitic gumma of, 333
tuberculous infiltrations in, in childhood (abs), 411
Luxation, coxofemoral congenital, roentgen sign (abs), 427
Lymph nodes, roentgen study of (abs), 286
roentgen visualization in animals (abs), 139
Lymphatic system of thorax and diaphragm, roentgen study of absorption by, of thorium dioxide injected intrapleurally into animals, 166
roentgen study of (abs), 286
Lymphatics, cervical, management of cancer of, 366
Lymphogranulomatosis, malignant: See Hodgkin's disease

Mastoids, roentgen examination, 482
technical points in roentgenography of, 106
Mediastinum, tumor of, treated by radiotherapy (abs), 575
tumors of, roentgen diagnosis and differentiation, 458
Melville, Stanley, obituary notice (E), 680
portrait, 680
Menopause, surgical, sympathetic functional radiotherapy in (abs), 579
Mesenterium commune (abs), 421
anomaly, in connection with a case complicated by appendicitis (abs), 563
Metabolism, mineral, effect of roentgen irradiation on (abs), 580
Metastases, osseous, in cancer of breast and uterus (abs), 566
Metatarsal, second, fracture (abs), 428
bones, marching fractures of (abs), 712
bones, short, and their relation to poliomyelitis (abs), 710
Metatarsus varus, congenital (abs), 709
Michigan Association of Roentgenologists, 262
Minnesota Radiological Society, 547
Mouth, management of cancer of, and cervical lymphatics, 366
Muscles, skeletal, benign angiomatous tumors of (abs), 711
Myelography, with iodized oils, dangers, inconveniences and injuries (abs), 565
with small amounts of contrast medium (abs), 125
Myeloma, multiple, aleukemic and leukemic (abs), 139
multiple, of vertebral column (abs), 426
multiple, with blood picture of plasma cell leukemia (abs), 138
Myelosis, conversion of chronic into acute leukemic (abs), 571
Myocardium, calcification in, 766

Nasopharynx, fibromas, treatment by some physical agents (abs), 574
Neoplasms: See Tumors

NEPHROPYELOGRAPHY, its clinical value (abs), 122
results obtained with uroselectan and abrodil (abs), 123
NEUROBLASTOMA, radiosensitive, 234
NEW-BORN, non-rachitic cupping of long bones in (abs), 136

OCCULT TAIL, case of (abs), 569
OLECRANON, tuberculosis of (abs), 134
OPHTHALMOLOGY, borderline rays in (abs), 573
OS ACETABULI, 492
OSSIFICATION of soft parts of shoulder (abs), 567
roentgen observations on disturbances of, in borders of vertebrae (abs), 137
OSTEITIS deformans (Paget's) in relation to cardiovascular disease (abs), 705
deformans, roentgen signs (abs), 426
fibrosa, generalized, improved after removal of parathyroid tumor (abs), 425
fibrosa cystica, roentgen evidence of extensive, calcification of kidneys in, 593
tuberculosa multiplex cystica (Jüngling) (abs), 703
OSTEOCHONDRITIS of internal cuneiform (abs), 711
OSTEOGENESIS imperfecta (abs), 566
imperfect congenital, identity of, and idiopathic osteopsathyrosis (abs), 136
OSTEOMYELITIS, chronic, carcinoma in, 787
OSTEOPHYTOSIS, generalized, clinical, roentgen and histopathological findings in case of (abs), 703
OSTEOPSATHYROSIS, clinical and roentgen picture of (abs), 566
idiopathic, differentiation of infantile scurvy from (abs), 136
OSTEOPOIKILOSIS, hereditary (abs), 426
OTTO'S PELVIS, 346
OVARIES, comparative histology of, in relation to question of injury of germ plasm by roentgen rays (abs), 714
possibility and probability of injury to germ cell after irradiation of (abs), 715

PACHYMENINGITIS after roentgen irradiation? (abs), 275
PAGET'S DISEASE in patient with large calcified fibromyoma (abs), 134
PANCREAS, roentgen therapy of, in treatment of asthma (abs), 576
PARATHORMONE, treatment of megacolon with (abs), 421
PARATHYROID GLANDS, roentgen studies of deossification, 496
rôle of, in diseases associated with demineralization of human skeleton (abs), 704
tumor, case of generalized osteitis fibrosa improved after removal of (abs), 425
PARATHYROIDECTOMY, considerations on use of, for arthritis (abs), 708
PARATHYROIDISM, certain arthritic disturbances associated with (abs), 708
in reference to orthopedic surgery (abs), 705
PARENCHYMATOUS ORGANS, roentgen opacification of, by intravenous injection of thorotrast (abs), 285
PATELLA, roentgen symptomatology of (abs), 134
unusual anomaly of (abs), 570
PELLEGRINI-STIEDA'S DISEASE (abs), 570
PEPTIC ULCER, chronic, of traumatic origin (abs), 560
gastroscopic study of (abs), 560
pathogenesis of perigastric abscess complicating (abs), 420
roentgen appearance of associated niches in ulcus ventriculi (abs), 560
roentgen diagnosis of postoperative jejunal ulcer and of gastrojejunocolic fistula confirmed by operation (abs), 562
ulcerus duodenal bulb (abs), 561
unrecognized, barium meal examination of stomach in presence of, 191
PERICARDIUM, diverticula (abs), 413
PHARYNX, highly malignant tumors of (abs), 719
pouches of (abs), 409
pouches of, diagnostic importance of filling (abs), 696
PHRENICOEXAIRESIS, left-sided, fluttering of abdominal viscera as a complication following (abs), 119
PHYSIOLOGY, gastric, mechanical factors of, 735, 745
PITUITARY GLAND, basophil adenomas of (abs), 408
tumors, roentgen diagnosis (abs), 695
PLACENTA, roentgen visualization, 37
roentgenography (abs), 120
previa, roentgen diagnosis, 230
PNEUMOCEPHALUS, traumatic (abs), 408
PNEUMONIA, due to aspiration of lipoids (abs), 412
experimental tuberculous, resolution of, 721
PNEUMOTHORAX, spontaneous bilateral (abs), 410
spontaneous, in an infant, 770
POLIOMYELITIS, acute anterior and the Bordier method (abs), 575
relation of short metatarsal bones to (abs), 710
POLYPOSIS, intestinal (abs), 422
POLYPS, gastric, and pernicious anemia syndrome (abs), 560
PROSTATE GLAND, benign hypertrophy, roentgen treatment, 350
roentgen exploration of (abs), 121
PROTRACTED FRACTIONAL METHOD of irradiation: See IRRADIATION and ROENTGEN THERAPY
PYELOGRAPHY: See also NEPHROPYELOGRAPHY, URETEROPYELOGRAPHY and UROGRAPHY
application of syncholia and synuria of certain iodized compounds (abs), 430
ascending and descending, comparative morphological study of kidneys and ureters (abs), 124
excretion (abs), 124
intravenous, in kidney tuberculosis (abs), 125
intravenous, in urinary bilharziasis, 208

intravenous, with abrodil (abs), 123
Pyelo-ureteritis cystica, 778
Pyopneumocyst, roentgen diagnosis (abs), 418

Radiation, combination of roentgen and light (abs), 276
fractional administration of large amounts of, skin erythema course in (abs), 286
poisoning, chronic occupational (abs), 283
relation of, to genetics and mutation theory of cancer (E), 252
therapy, early experiences in, 153
Radiologic service in the United States (E), 387
Radiology, relation of American College of Radiology to future of, 164
Radium, celluloid as support in application of (abs), 283
needle carrier, 109
needles, long, determination of dosage for, 74
D, E and F, action of, with colloidal bismuth upon growing tumors (abs), 284
E, localization of, associated with bismuth after injection in cancerous animal (abs), 283
salts and emanation, 830
simple detector for, 689
Radius, fractures of inferior extremity of, reduction under screen (abs), 423
Radon, ingestion of, and its possible health dangers, 673
needles, long, determination of dosage for, 74
Reticulo-endothelial system, question of blockage of (abs), 282
Ribs, lumbar (abs), 567
Rickets, and renal dwarfism (abs), 424
Roentgen examination repeated after thirty years, 44
films, instrument for measuring density of, 550
ray absorption of tooth slabs, quantitative method for studying, 663
ray standards and units, 815
rays, comparative histology of ovaries in relation to question of injury of germ plasm by (abs), 714
rays, late injury by, and consequences for radiotherapy (abs), 713
therapy, data concerning three years' experience with 600 kv. (peak), 520
therapy, present status of, with voltages above 200 kv., 628
therapy, protracted fractional, of malignant tumors, results (abs), 143
tube with internal radiation protection for 400 kv. operation voltage (abs), 577
tubes, high voltage, comparison of, 378
Roentgenograms, box for viewing when wet, 398
how should they be viewed? (abs), 283
of chest taken with breath held in expiration, 266
of chest, triangular basal shadows in (E), 838
pulmonary, device for taking, under control of heart beat (abs), 143
Roentgenographic exposure, improved device for synchronizing, with patient's pulse, 104
Roentgenography, frame for holding infants upright during, 396
oblique view, for demonstration of articular facets in lumbosacral backache and sciatic pain, 173
Roentgenoscopy, value of method of adaptation of one eye (abs), 287

Sacrum, paraglenoid sulci of (abs), 569
transverse fracture of, 676
Sarcoma, osteogenic (abs), 126
primary, of vertebrae (abs), 126
transplantable mouse sarcoma No. 180, reaction of, to radiations of different wave length, 614
Scaphoiditis, tarsal, in young children (abs), 427
Scoliosis, nephrogenic (abs), 120
Scurvy, infantile, differentiation of, from idiopathic osteopsathyrosis (abs), 136
Sedimentation reaction of red blood corpuscles immediately before and after roentgen irradiation (abs), 281
Sella turcica, deformation of, in tumors of middle cranial fossa, 23
Shoulder, ossification of soft parts of (abs), 567
Sinus, sphenoidal, some experiments in projection of, through orbit (abs), 143
Sinuses, technical points in roentgenography of, 106
Skiodan: See Abrodil
Skull, deformation of sella turcica in tumors of middle cranial fossa, 23
occipital view, modified technique, 271
sutures of, interpretation of roentgen picture (abs), 558
Spasm of cecocolic sphincter (abs), 700
Spina bifida of atlas and axis (abs), 701
Spine, calcareous deposits in intervertebral discs (abs), 703
case of complete dislocation between fifth and sixth cervical vertebrae without fracture (abs), 701
cervical, form of spondylarthropathy of, with radicular disturbances (abs), 702
cervical, traumatic subluxation (abs), 567
changes in, following tetanus (abs), 703
costovertebral arthritis (abs), 136
fractures, results (abs), 425
horizontal thickening within vertebral bodies (abs), 568
multiple myeloma (abs), 426
normal and pathologic anatomy of contours of vertebral bodies (abs), 702
normal and pathological physiology of nucleus pulposus of intervertebral disc (abs), 568
oblique view for demonstration of the articular facets in lumbosacral backache and sciatic pain, 173
persistent apophyses of (abs), 702

roentgenography of, 273
spina bifida of atlas and axis (abs), 701
SPLEEN, hemangiosarcoma of, changes in bones (abs), 134
histological studies of, in rabbits following intravenous injection of thorium dioxide, 512
SPONDYLARTHROPATHY of cervical spine with radicular disturbances (abs), 702
STANDARDIZATION service of ionometers at St. Antoine Hospital (abs), 287
STASIS, intestinal, in infants and children (abs), 421
STENOSIS, syphilitic, in digestive tract (abs), 118
STERILIZATION, temporary, by irradiation, and research in human heredity (abs), 712
temporary, from irradiation, disputed questions in regard to (abs), 712
temporary, problems of dosage in, by irradiation (abs), 714
temporary roentgen, clinical experience (abs), 714
temporary roentgen, estrus as sign of beginning of ovulation after (abs), 715
STERNUM, bifide (abs), 567
STOMACH, barium meal examination of, in presence of unrecognized perforated peptic ulcer, 191
benign tumors of (abs), 419
incidence of carcinoma in certain chronic ulcerating lesions of (abs), 419
intrathoracic congenital, in child aged seven (abs), 559
pointed diverticula of fundus of (abs), 700
polyps of, and pernicious anemia syndrome (abs), 560
some mechanical factors in physiology of, 735, 745
SUBLUXATION, traumatic, of cervical spine (abs), 567
SULCI, paraglenoid, of ilium and sacrum (abs), 569
SUPRARENAL GLANDS, action of radiotherapy in region of, on constitution of osseous callus in old fractures (abs), 432
arteritis obliterans treated by roentgen therapy of region of (abs), 576
calcification of, in Addison's disease (E), 93
calcification of, in tuberculosis of (abs), 420
radiotherapy of region of, in gangrene and diabetic pruritus (abs), 575
treatment of exophthalmic goiter by irradiation of region of (abs), 576
SYMPATHETIC SYSTEM, radiotherapy of, in pernicious vomiting (abs), 432
radiotherapy of, in surgical menopause (abs), 579
SYPHILIS of lung, 333; (abs), 410
stenoses from, in digestive tract (abs), 118
tertiary, of the esophagus, 773
SYRINGOMYELIA, roentgen treatment (abs), 141

TECHNIQUE, hard rays and rapid exposure time in pulmonary teleroentgenography (abs), 144
occipital view, modified technique, 271
roentgen examination in the position "feet toward the ceiling" (abs), 283
roentgenography of spine, 273
some experiments in projection of sphenoidal sinus through orbit (abs), 143
use of chest roentgenograms taken with breath held in expiration, 266
TELEROENTGENOGRAPHY, hard rays and rapid exposure in pulmonary teleroentgenography (abs), 144
pulmonary, Rotalix tube in (abs), 287
TENDINITIS OSSIFICANS TRAUMATICA (abs), 570
TENDO PATELLAE, roentgen consideration of its length (abs), 570
TENEBRYL, new substance for intravenous urography (abs), 123
TERATOMA TESTIS, diagnosis and treatment, 356
TETANUS, changes in spine following (abs), 703
THORAX: See CHEST
THORIUM DIOXIDE, biological observations on storage of—phagocytosis (abs), 282
histological studies of liver, spleen and bone marrow in rabbits following intravenous injection of, 512
roentgen study of absorption by lymphatics of thorax and diaphragm of thorium dioxide injected intrapleurally into animals, 166
THOROTRAST, and infection (abs), 282
experimental researches on roentgen opacification of various parenchymatous organs by intravenous injection of (abs), 285
study of lymphatic circulation and lymph nodes by injection of (abs), 286
use in diagnosis of intra-abdominal tumors (abs), 119
THYROID GLAND, malignant tumor, results of irradiation in two cases (abs), 278
TISSUES, cultivation of, in plasma of animals that have been irradiated with roentgen rays (abs), 579
normal, stimulant action of roentgen rays on (abs), 580
TONGUE, base of, highly malignant tumors of (abs), 719
TONSILS, carcinoma, review of 122 cases (abs), 177
diagnosis and treatment of malignant conditions of (abs), 718
TOOTH SLABS, quantitative method for studying roentgen-ray absorption of, 663
TRACHEA, displacement of, as roentgen sign of congenital cardiac disease in infants, 470
TRICUSPID DISEASE, roentgen findings (abs), 413
TROCHANTER, great, new method of determining presence of upward displacement of (abs), 569
lesser, of femur, unusual changes in, 224
TUBERCULOMA of small intestine (abs), 563
TUBERCULOSIS, bone, two peculiar cases of (abs), 135
early, historical review of question of roentgen demonstration of cavities caused by de-

struction of lungs in (abs), 697
hematogenous (non-miliary) pulmonary, 442
of kidney, intravenous pyelography in (abs), 125
of large bowel, surgical treatment (abs), 700
of olecranon (abs), 134
of suprarenal glands, calcification in, roentgen study in Addison's disease (abs), 420
pulmonary, bronchography as an essential and safe adjunct in study of, 301
pulmonary, bronchography in relation to pathology of, 308
pulmonary, chronic, bronchography in amplification of roentgen film in, 289
pulmonary, in childhood, 16; (abs), 411
pulmonary, round foci type of, 319
Tumor, malignant, of thyroid, results of irradiation in two cases (abs), 278
mediastinal, treated by radiotherapy (abs), 575
of cecum simulating ileocecocolic invagination (abs), 116
of ileum causing recurrent invagination (abs), 116
Tumors, benign angiomatous, of skeletal muscles (abs), 711
benign, of stomach (abs), 419
cerebral, ameliorated by radiotherapy (abs), 572
growing, effect of radium and bismuth on (abs), 284
hypophyseal, roentgen diagnosis (abs), 695
intra-abdominal, use of thorotrast in diagnosis of (abs), 119
intracavitary, direct roentgen irradiation, 64
intracavitary, treatment, 394
lympho-epithelial (abs), 278
malignant, of pharynx and base of tongue (abs), 719
malignant, protracted fractional irradiation of (abs), 144
malignant, protracted fractional roentgen therapy of, results (abs), 143
malignant, relations between radiosensitiveness of, and mother tissue (abs), 716
mediastinal, roentgen diagnosis, and differentiation, 458
of embryonic structure—embryocytomas—and their radiosensitiveness (abs), 716
of heart (abs), 413
of middle cranial fossa, deformation of sella turcica in, 23
of small intestine, roentgen diagnosis (abs), 420
radiosensitive, of blood forming organs, urologic aspects (abs), 423

Ulcer, gastric, See Peptic ulcer
Umbrenal, use in pyelography (abs), 124
Uremia, digestive, errors of diagnosis in course of (abs), 119
Ureterocele, recognition of (abs), 423
Ureteropyelography, retrograde, in hydronephrosis (abs), 122
Urethra, female, studies of, especially as regards closing mechanism of bladder (abs), 120
male, roentgen exploration of, and of prostate (abs), 121
Urethrography in the male (abs), 121
Urinary apparatus, roentgen examination of, by means of excretion of contrast-giving substances (abs), 124
Urography, intravenous, by means of tenebryl (abs), 123
Uroselectan used in pyelography (abs), 123; (abs), 124; (abs), 125
Uterus, cervix and vagina, carcinoma, statistics of for years 1923–1925 (abs), 142
cervix, cancer, results of operation and irradiation (abs), 719
cervix, carcinoma, problems in radiation therapy of, 60
cervix, carcinoma, treatment (abs), 281
determination of position of portio and uterus in gynecological roentgen therapy (abs), 280
fibromyoma, irradiation in treatment, 51

Varicose disease of legs, roentgen observations (abs), 138
veins, "resting infection" in, diagnosis and treatment (abs), 428
Vertebrae, abscess of, unusual migration of (abs), 138
primary sarcoma of (abs), 126
roentgen observations on disturbances of ossification in borders of (abs), 137
Vomiting, pernicious, and sympathetic radiotherapy (abs), 432
Vulva, carcinoma, results of irradiation (abs), 142

Wave length, variations in dosage dependent on, 538

AUTHOR INDEX TO VOLUME XXXI

Adler, Karl, 580
Afifi, Mahmoud Ahmed, 208
Åkerlund, Åke, 420
Akerman, J., 425
Alajouanine, Th., 427
Albrecht, Hans, 714
Allen, M. Lowry, 470
Allen, William E., Jr., 676
Allison, R. G., 16
Altman, M., 415
Altschul, Walter, 277, 393
Ané, J. N., 139, 166
Antonucci, Cesar, 415
Arce, Francisco, 265
Arce, Manuel, 265
Arndt, Julian, 279
Arntzen, Leif, 116
Augier, Pierre, 422

Baastrup, Chr. I., 138
Ball, Ralph G., 420
Ballin, Max, 705
Barber, T. H. T., 138
Barker, Howard H., 673
Barney, J. D., 423
Barr, David P., 429
Barringer, Benjamin S., 350, 721
Bársony, T., 700
Bauer, Walter, 706
Bayer, Ludwig, 287
Bayley, Robert H., 414
Beau, H., 431
Becchini, Gastone, 118
Béclère, 120
Béclère, Henri, 283
Behnken, H., 815
Bendove, Raphael A., 323
Benhamou, Ed., 417
Bensaude, Raoul, 422
Bergk, K., 143
Betocchi, Giorgio, 558
Bianchini, Alessandro, 120, 124
Bidoli, Emilio, 136
Bieberbach, Walter D., 778
Bienvenue, A., 579
Bierdermann, Fritz, 700
Bigler, John A., 565
Bignami, G., 140
Bishop, Francis W., 663
Bishop, Paul A., 1
Blackford L. Minor, 414
Blomström, H., 415
Böhm, Alexander, 276
Bogart, Franklin B., 709
Borak, J., 715, 716
Bosch, Hans, 579
Bouslog, John S., 410
Bouton, E., 287
Bowen, David R., 547
Bradford, W. L., 571
Brancovici, M., 575
Braun, H., 280
Braun, R., 264
Brdiczka, I. G., 409, 560
Broders, Albert C., 719
Brooksher, W. R., Jr., 117, 122
Brown, C. E., 698
Brown, Samuel, 41
Bruck, Samuel, 319
Brumbaugh, Herbert L., 570
Brunet, 576
Buchman, Joseph, 711
Buckstein, Jacob, 100
Bucky, G., 282
Buisson, Mario, 559
Buisson, Paolo, 115, 122
Burlingame, R. M., 135
Burman, Michael S., 224
Burnam, Curtis F., 718
Burt, Edward F., 136
Buschbeck Herbert, 709
Butler, Carlos, 716

Calderón, Hector Ramirez, 566
Calo, A., 579
Camp, John D., 420
Capizzano, Nicolas, 573
Cappelli, Luigi, 283
Capua, Antonio, 563
Cardinale, G. B., 549
Carulla, Vincente, 578
Casati, Annibale, 144
Chantraine, H., 143
Chasin, Adib, 703
Chavany, 576
Chéne, Paul, 560
Chérigié, 576
Chievitz, Ole, 425
Childrey, John H., 719
Christiani, Edmund, 281
Christie, Arthur C., 389
Christopher, Frederick, 567
Clark, D. M., 541
Clark, Dwight F., 710
Codet, P. H., 567
Coliez, Robert, 573
Collins, J. J., 787
Compere, Edward L., 568, 704
Cone, Sydney M., 704
Cook, Philip H., 778
Cordonnier, V., 432
Correra, Tommaso, 566
Corrigan, K. E., 628
Costolow, William E., 520
Cottenot, 576
Cushing, Harvey, 408
Cutler, Max, 90, 720, 819

D'Aubigné, Merle, 423
D'Emidio, Angelo Santoro, 574
Dachtler, H. W., 394
Davenport, Tom F., 414
Davidoff, Leo M., 696
de Lorimier, Alfred A., 496
de Takats, G., 428
Dean, Archie L. Jr., 350
del Buono, Pietro, 134
del Guidice, Vincente, 578
Delaney, P. Arthur, 711
Delario, A. J., 177
Delherm, 576
Delherm, L., 431
Desaunay, A., 579
Desjardins, A. U., 255
Desplats, R., 432, 575
Determann, A., 413
di Bella Filippo, 126
Didiée, 570
Didiée, Jean, 134
Drevon, 427
Druckmann, Adolf, 715
Duffy, James J., 350, 717
Dufour, Pierre, 567
Dupas, C., 432
Dwyer, Hugh L., 424
Dyke, Cornelius G., 696
Dyroff, Rudolf, 714

Edeiken, Joseph, 414
Edling, Lars, 125
Ehrenburg, G. E., 119
v. Eiselberg, A., 408
Eliasz, Ernst, 699
Elzberg, Charles A., 695
Emery, Clyde K., 520
Enderle, C., 565
Engelstad, Rolf B., 118
Ettinger, Alice, 593
Eversbusch, G., 698
Ewing, James, 97, 153

Faber, Alexander, 709
Farrell, John T., Jr., 136
Feissly, R., 430
Feldman, Maurice, 227
Felsen, Joseph, 340
Ferguson, Albert B., 710
Ferguson, Russell S., 356
Fetzer, H., 119, 413
Finsen, V., 135
Fischgold, 567
Fleischner, Felix, 421

Flinn, Frederick B., 830
Freedman, Eugene, 333, 492
Freedman, John, 410
Friedman, Lewis J., 349
Fumarola, G., 565
Funsten, Robert V., 708
Funston, Robert V., 135

Galifi, Liborio, 116
Gallart Monés, F., 101
Gallavresi, Luigi, 117
Garcia-Calderon, J., 121
Gass, R. S., 412
Geipel, P., 701
Gejrot, W., 423
George, Arial W., 433
Gernez, L., 574
Gershwin, Benjamin S., 323
Gerstel, Gustav, 703
Geschickter, Charles F., 126
Geyman, M. J., 541
Ghormley, Ralph K., 173
Gianturco, Cesare, 735, 745
Gibert Queralto, J., 578
Giese, E., 141
Gilbert, R., 118, 418, 430
Ginsburg, Theodore M., 429
Giraudi, Gioffredo, 569
Glass, Ameil, 328
Glasser, Otto, 686
Gloor, W., 279
Goette, K., 702
Goia, Ioan, 431
Goldmark, Carl, 409
Goltz, Hilda Lee, 109
Goodale, Raymond H., 778
Gorjelik, R., 722
Gottlieb, Filip, 427
Gouin, J., 579
Gowen, G., 703
Graham, Evarts A., 145
Greene, Carl H., 420
Grinker, Roy R., 687
Grossmann, G., 576
Gudjonsson, Sk. V., 125
Guénaux, 559
Gunsett, A., 287, 577
Gutmann, René A., 561
Guttman, Paul H., 200
Györgyi, Géza, 696

Haenisch, F., 124
Haglund, Patrik, 428
Hakansson, Eric G., 421
Hampton, Aubrey O., 419
Hângănut, M., 561
Haring, Wilhelm, 560, 699
Hartung, Adolph, 410
Hatieganu, I., 561
Hauser, Harry, 234
v. Hayek, Heinrich, 567
Hayer, E., 722
Heald, Alfred H., 44
Healy, William P., 60
Heise, Agnete, 136
Held, A., 282
Hellmer, Hans, 137
Hellström, John, 423
Henny, George C., 550
Herendeen, Ralph E., 350
Hernaman-Johnson, F., 143
Heuser, Carlos, 120
Higley, Charles S., 333
Hillemand, Pierre, 422
Hillstrom, H. T., 412
Himler, L. E., 784
Hodge, Harold C., 663
Hodges, Fred M., 238
Hodges, Paul C., 399
Hoffmann, Carl, 278
Holmes, George W., 419
Holthusen, H., 140, 264
Holzmann, Max, 413
Hoppe, G., 119
Horsters, Hans, 699
Hoyne, Archibald, 565
Hudson, C. L., 31
Huet, J.-A., 119
Hunter, F. T., 423

Iaobovici, I., 426
Imboden, Harry M., 419
Impiombato, Gastone, 563
Ingvar, Sven, 125
Israelski, Martin, 564

Jacobaeus, H. C., 412
Jäderholm, K. Bruno, 413
Jannson, Gösta, 422
Jenkins, Hilger P., 711
Johannesson, Carl J., 394
Johnston, C. G., 698
Joly, Marcel, 286
Jones, Thomas E., 281
Junet, R., 284

Kadrnka, S., 118, 122, 284, 564
Kantor, John L., 751
Kaplan, Ira I., 100
Karelitz, Samuel, 424
Kato, Katsuji, 712
Kaye, G. W. C., 815
Keller, A. E., 412
v. Kémeri, D., 140
Kerley, Charles G., 421
Kern, Richard A., 421
Keyes, Donald C., 568
Kirklin, B. R., 173, 259, 581
Knittel, Gottlob, 421, 565
Koch, Lewis A., 571
Körbler, Juraj, 282
Kolomoyseff, H., 424
Koppenstein, E., 700
Kornblum, Karl, 23
Korol, Ephraim, 266
Kraft, Ernst, 712
Krasso, 573
v. Krenninger-Guggenberger, J., 715
Kröker, Peter, 563
Krumreich, Robert, 282
Kühne, Konrad, 703

Lacassagne, A., 283
Langeron, L., 432, 575
Lapidus, Paul W., 570
Laurell, Hugo, 116
Law, Frederick M., 263, 482
Lazeanu, E., 575
Lazeanu, G., 565
Le Page, 144
Ledbetter, L. H., 106
Leddy, E. T., 544
Ledoux-Lebard, R., 121, 283, 549
Legueu, Fey, 123
Leitner, Z., 282
Leonard, Ralph D., 433
Lepennetier, F., 415
Leucutia, T., 628
Levene, George, 398, 554, 588
Levin, Alfred G., 204
Levin, Isaac, 280
Levine, Samuel, 487
Liberson, F., 690
Liwschitz, S., 722
Lockwood, Bruce C., 419
Lönnerbald, Lars, 115
Lorey, A., 275
Loughery, Thomas P., 64
Lovisatti, Nicola, 118
Lowenburg, Harry, 429
Lupacciolu, Giovanni, 562
Luxenburger, Hans, 712
Lyon, Ernst, 426, 568

MacMillan, A. S., 409
McNaughton, Elizabeth, 138
Magendantz, Heinz, 593
Maino, Mario, 549
Major, S. G., 700
Mallet, Lucien, 574
Marchioni, 559
Marchioni, R., 417
Mardersteig, Klaus, 702
Markow, D. A., 722
Martin, Ann, 411
Mathieu, Pierre, 572
Matthews, Helen, 398, 554, 588
Maurer, E., 713
Mauric, G., 427

Mayer, Ernst G., 695
Medelman, J. P., 16
Meland, Orville M., 520
Menville, Leon J., 139, 166
Meyer, J., 577
Migliardi, Luigi, 125
Mintz, E. R., 423
Møller, P. Flemming, 125
Montgomery, Albert H., 558
Moore, John J., 496, 766
Moragues Gonzalez, P., 578
Moschetta, Giovanni, 117
Mouchet, Albert, 427
Moutier, François, 560
Mudd, Seeley G., 520
Müller, Walther, 577
Muller, Guli Lindh, 138
Muro, Felipe, 709
Murphy, J. E., 301
Mutscheller, A., 244
Muzii, Mario, 116

Nachlas, I. William, 708
Neeff, Th. C., 714
Nemenow, M., 716
Nemours-Auguste, 123, 287, 417
Neuhof, Harold, 289
New, Gordon B., 719
Nichols, B. H , 346
Nicotra, Antonino, 558
Niedner, Franz, 702
Nielsen, Jens, 283
Nürnberger, L., 713
Nyka, W., 283

O'Neill, D. B., 104
Ogilvie, Robertson, 572
Olsen, Hans C , 425
Orley, A., 138
Orr, H. Winnett, 423
Ossinskaja, W., 141

Pacini, Danilo, 134
Pape, R., 698
Paschetta, Vincent, 570
Peabody, Charles W., 709
Peirce, E. G., 137
Pellini, Maria, 115
Pemberton, Ralph, 137
Pendergrass, Eugene P., 470
Perussia, Felice, 686
Petetin, J., 121
Petrignani, Roger, 123
Pfahler, George E., 51, 458
Philipp, E . 142
Pierron, E., 101
Piersol, George M., 428
Pierson, John W , 412
Pigorini, Luigi, 563
Piney, A., 139, 571, 572
Pinner, Max, 442
Pinós, T. A., 101
Piot, Etienne, 562
Ployé, 567
Pohl, R., 421, 697
Pohle, Ernst A., 512
Ponzio, M., 276
Popp, L., 413, 563
Portmann, U. V., 46
Potter, B. P., 308
Powell, Clilian B., 37
Pugno-Vanoni, Enzo, 686

Quick, Douglas, 366
Quimby, Edith H., 74, 90, 91
Quint, H. A., 710

Rachlin, Nathan H., 569
Ramond, Felix, 560
Rankin, Fred W., 700
Rathcke, L., 703
Rawak, F., 696
Raynal, Albert, 574
Reed, Edward N., 701
Reed, Jewett V., 408
Reinhard, M. C., 538
Reisner, Alfred, 286
Reitter, George S., 770
Reynolds, Lawrence, 93, 678, 838
Reynolds, Russell J., 569
Riach, Jean S., 139, 572
Rieder, H., 697
Ritchie, Gorton, 512
Robertson, Robert C., 709
Rochlin, D. G., 424
Rodgers, Mortimer, 701
Roesler, Hugo, 1
Roffo, A. H., 574
Ronneaux, Georges, 575
Rosi, Peter A., 420
Rowntree, Leonard G., 420
Ryan, William T., 566
Rypins, Edwin L., 597

St. Jianu, 426
Salinger, Hans, 697
Samek, Julius, 275
Sandera, Robert, 699
Sandström, Carl, 415, 416
Sante, L. R., 91
Santoro, Mario, 134
Sarasin, R., 564
Saul, Walter, 698
Schaltenbrand, G., 275
Schechter, Samuel, 751
Schinz, Hans R., 279
Schloss, Wilhelm, 277
Schmidt, Ernst A., 200
Schmitt, Walter, 719
Scholz, Thomas, 409, 792
Schoonover, Janetta Wright, 532
Schreus, H., 723
Schröder, Robert, 141
Scott, Harry A., 266
Seifert, E., 580
Shapiro, Benjamin, 571
Shattuck, Howard F., 419
Sheldon, Luther, 421
Shiels, Ethol Hall, 532
Shiflett, E. L., 346
Sichel, D., 287
Sickels, T. N., 31
Simon, Joseph, 418
Simon, Stefan, 142
Simonson, S. G., 424
Singer, G., 378
Singer, Harry A., 191, 420
Sluys, F., 430
Smith, E. Gerard, 823
Snoke, Paul O., 570
Snow, William, 37
Soiland, Albert, 90, 164
Solis-Cohen, Leon, 487
Solomon, 287
Solomon, I., 815
Sowerbutts, Joseph, 410
Speder, E., 432
Spitz, Leonhard, 421
Stähler, Fritz, 719
Stecher, William R., 64
Stein, Arthur, 701
Steiner, Georg, 278
Steinfield, Edward, 428
Steinitz, E., 276
Stewart-Harrison, R., 724
Stoneburner, C. F., 378
Stoppani, F., 286, 428
Stout, Arthur Purdy, 263
Straus, Francis Howe, 712
Strouzer, V. S., 567
Stuhl, 576
Sugiura, Kanematsu, 614
Sutherland, Charles G., 110
Svab, Vaclav, 426
Swanson, Leonard A., 139
Swenson, Paul C., 204

Taft, Robert B., 689
Taylor, A. G. C., 284
Taylor, Lauriston S., 378, 815
Teneff, S., 286
Thibonneau, M., 572
Thompson, Charles F., 408
Thomsen, Einar, 120
Tixier, Léon, 575
Truchot, 123
Tschakert, J., 409

Ude, Walter H., 230
Unseld, E., 721

Unshelm, E., 411
Urner, J. A., 230

Van Alstyne, Guy S., 703
Van Huysen, Grant, 663
Van Leeuwen, W. S., 409
Van Pee, P., 575
Vasselle, 559
Vastine, Jacob H., 51, 603
Velde, G., 697
Viallet, 559

Waggoner, R. W., 784
Warren, S. Reid, Jr., 104
Warren, Stafford L., 663
Wasson, W. Walter, 410
Waters, Charles A., 100
Weber, Harry M., 607
Weltz, G. A., 409, 698
Westermark, Nils, 412
Weum, T. W., 230
Weyl, Charles, 104
Wheatley, Frank E., 398, 554, 588
Whipple, G. H., 571
Whitmore, W. H., 271, 273
Widmann, Bernard P., 532
Wilcox, Leslie F., 773
Wilke, Adolf, 697
Willis, Henry Stuart, 721
Wilson, May G., 558
Windholz, Franz, 426
Wintz, H., 712
Wohlers, H., 122
Wolf, Artur, 698
Wolferth, Charles C., 414
Wood, Horatio C., Jr., 396

Zangheri, Carlo, 138
Zimmer, Theodor, 577
Zimmern, 576
Zuppinger, A., 143
Zwerg, H. G., 144